

SECOND EDITION

# AVOIDING COMMON ERRORS IN THE EMERGENCY DEPARTMENT

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Every attempt has been made to ensure the accuracy of management recommendations and medication dosages. However, the reader is urged to consult other resources for confirmation of recommendations and medication dosages.

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# PREFACE

Emergency medicine is high risk. Emergency care providers are tasked with caring for patients they don't know in a time-pressured and overcrowded environment. Complicating matters further is the fact that the majority of patients in the emergency department have non-life-threatening conditions, so health care providers are easily lulled into a false sense that the patient laying before them has a benign condition. The great challenge in emergency medicine is to sort through the morass of benign conditions in order to properly find and treat the deadly ones to find the proverbial needles in the haystack. Even when confronted with non-life-threatening conditions, acute care providers face the challenge of delivering excellent care efficiently, communicating with patients quickly, discharging patients with proper outpatient care instructions, and then rapidly moving on to the next patient. Errors in this setting are inevitable. It is our goal in creating this textbook to help minimize those errors by educating the reader about common mistakes and pitfalls in the practice of emergency medicine.

Most second edition textbooks tend to be fairly similar to prior editions, generally with only 15% to 20% turnover of topics and writers. For this second edition, however, we decided to take a completely different approach. The majority of authors to this second edition are new, and those that are repeat authors were given completely new topics on which to write. We intentionally did this to provide a large group of new topics as well as a fresh look at repeat topics. The result is that this new text reads more like a second *volume* rather than a second *edition*. We anticipate that readers will continue to benefit from the first edition even as they delve into the second.

As with the first edition, we chose topics based on three main themes: errors that are common, errors that pose imminent danger to the patient, and errors that pose significant medicolegal risk to the health care provider. The majority of errors that we've included have been organized into various organ systems, such as the cardiovascular system, neurologic system, and so on. In addition, separate sections are present, which focus on nonclinical aspects of emergency medicine practice such as proper documentation, communication with consultants, interactions with lawyers, etc. It is our expectation that that the text will be read not in a single sitting but instead will be savored over a much longer time period. Our choice of 365 chapters is no coincidence...we suggest that readers simply read one chapter per day

over the course of a year.

We'd like to thank the authors and associate editors for the significant time and effort that they devoted to making their chapters informative, cutting-edge, and practical. We thank the *Errors* Series Editor Dr. Lisa Marcucci for giving us the opportunity to create what we believe is an important educational contribution to the specialty. We also thank the publisher and managing editors at Wolters Kluwer for their support of this work. Finally, on behalf of the all of the authors and editors, we'd like to thank our families and colleagues for providing the inspiration to carry on our work.

It is our sincere hope that the reader finds this text practical and usable and that learning about these common errors produces a tangible improvement in patient care. Our best wishes to you and your patients!

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**Michael E. Winters, MD**

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—Amal Mattu, MD

This book is dedicated to Karen, my beloved wife; to Sydney, William, and Nathan who have made me appreciate the really important things in life; to my parents who fostered a spirit of growth; and to the residents and students of emergency medicine who help us all to remember the joy of learning and who reinforce a commitment to one of the greatest specialties.

—Arjun Chanmugam, MD, MBA

To Joyce, Moses and the residents, students, and patients of the Los Angeles County/University of Southern California Medical Center.

—Stuart Swadron, MD, FRCPC

I would like to thank my parents, Brenda and Joe, for believing in me; my family, Michelle, Anna, and Garrett, for their patience with me; and my residents in the combined Emergency Medicine and Pediatrics residency program for all the inspiration.

—Dale P. Woolridge, MD, PhD

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—Michael E. Winters, MD

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# SECTION I

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# CRASHING PATIENT

# **DON'T LOSE THAT AIRWAY!**

## **IMMINENT AIRWAY LOSS: WHO NEEDS ENDOTRACHEAL INTUBATION?**

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**NICHOLAS SAUBER, MD, RN-BSN, EMT-B AND DENA  
REITER, MD**

Airway assessment is arguably the first step in evaluating every patient in the emergency department (ED) setting. We will briefly discuss the assessment of a patient's airway and respiratory status, including clinical features that suggest that further respiratory support and airway management are required.

We perform endotracheal intubation in the ED for several reasons. In a broad sense, there are four indications to intubate: failure to protect the airway, failure to ventilate, failure to oxygenate, and anticipated clinical course. The emergency physician must be adept at the procedure of endotracheal intubation. In 2011, Walls et al. demonstrated that the vast majority of ED intubations are performed by emergency physicians, most commonly for medical indications, and with rapid sequence intubation being the most common method.

## **AIRWAY CONCERNS**

The most immediately life-threatening airway emergency is obstruction. Physical obstruction can result from foreign body, traumatic injury, penetrating injury, compression from a neck mass, expanding hematoma, or angioedema. Patients who have altered mental status may also have difficulty

managing secretions or may not protect their airway after an episode of emesis. In addition, even alert patients with copious secretions (e.g., bronchorrhea secondary to cholinergic toxicity) or severe hematemesis may not be able to safely protect their airway on their own. The airway must be immediately secured when any of these conditions are present, and a delay can have deleterious consequences.

When it is unclear whether or not the airway is at risk, clinicians may choose to use the Glasgow Coma Scale (GCS) to help aid in the decision to intubate. A GCS < 8 is considered comatose. However, it should be noted that patients may be at risk of airway complications at higher GCS scores. The gag reflex is also an unreliable indicator; the presence of a gag reflex does not ensure airway protection, and a gag reflex may be absent in healthy subjects.

## **VENTILATION AND OXYGENATION**

Ventilation broadly involves the ability to move air in and out of the lungs and allows for gas exchange at the alveolar level. Constricted bronchi and bronchioles secondary to a reactive process, asthma, or chronic obstructive pulmonary disease increase the work of breathing. Pulmonary edema, toxidromes, pneumonia, sepsis, acidosis, fever, or any other process that results in increased respiratory rate will also increase the work of breathing. When a patient cannot ventilate with the diaphragm alone, he will recruit accessory muscles to aid in inspiration and increase the respiratory rate. As these muscles fatigue, the patient will lose the ability to ventilate adequately. This in turn causes the partial pressure of carbon dioxide ( $\text{PaCO}_2$ ) to rise as gas exchange is compromised. Caution should be taken in interpreting  $\text{PaCO}_2$  levels from a blood gas, as a tachypneic patient may have low  $\text{PaCO}_2$  levels initially and a normal  $\text{PaCO}_2$  level may be the first signs of impending respiratory failure. Blood carbon dioxide levels above baseline cause respiratory acidosis and depressed mental status, further worsening the overall respiratory function and leading to rapid respiratory failure. Closely monitoring basic vital signs including pulse oximetry and continuous capnometry are crucial to early identification of ventilatory failure. When noninvasive methods are inadequate, endotracheal intubation with mechanical ventilation is the appropriate intervention.

Oxygenation is the other side of the same coin. A patient can be ventilating well but failing to oxygenate, typically due to an impaired diffusion of oxygen across the alveolar and capillary interface or an inability of the hemoglobin to bind oxygen molecules. Impaired diffusion occurs for

many reasons including cardiogenic pulmonary edema, pneumonia, aspiration, acute respiratory distress syndrome, inflammation, fibrosis, pulmonary embolism, or traumatic injury. Impaired heme oxygen binding is less common but seen in carbon monoxide exposure, acidemia, and methemoglobinemia. The treatment of these underlying pathologies is not the focus of this chapter, but attempts to correct underlying pathology must be made. When oxygenation is impaired to the extent that it cannot be corrected safely with less invasive methods (supplemental oxygen via nasal cannula, face mask, nonrebreather, or noninvasive ventilation), endotracheal intubation is the next appropriate step. Once the airway is secured, the patient can be mechanically ventilated to optimize gas exchange.

## ANTICIPATED CLINICAL COURSE

Lastly, the emergency physician must try to predict the patient's clinical course. While the airway, oxygenation and ventilatory status may all be adequate at the moment, various disease processes may lead to clinical deterioration. For example, an airway with evidence of smoke inhalation may be patent on initial evaluation only to later become compromised. A patient with severe agitation who requires diagnostics or treatment may need to be heavily sedated and intubated for his or her own protection and in order to complete necessary tests. Transporting a patient to another facility via ambulance or helicopter is tenuous and it may be prudent to secure the airway beforehand. It is the responsibility of the emergency physician to determine whether or not early airway intervention is judicious.

The decision to secure the airway can be challenging, nebulous, and subject to scrutiny. However, one will never be faulted for attempting to secure the airway too soon. It is delaying the decision, which leads to regret.

### KEY POINTS

- The main indications for endotracheal intubation are failure to protect the airway, failure to ventilate, failure to oxygenate, and anticipated clinical course.
- Ventilation is the broad ability to move air and exchange gases, while oxygenation is the ability to move adequate oxygen across alveoli to perfuse the tissues.
- Patients may need endotracheal intubation further down their clinical course. It is prudent to intubate early in these cases.

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## PREOXYGENATION

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**MICHAEL R. EHMANN, MD, MPH, MS**

Airway management is among the foundational skills of all emergency physicians. Despite the frequency with which the emergency physician engages in airway management, there are significant risks associated with airway manipulation. All patients—pediatric and adult, medical, and traumatic—benefit from practices and protocols that mitigate these risks. One such practice is appropriate oxygenation of the patient during the pre- and peri-intubation periods to lower the likelihood of hypoxemia throughout the process of tracheal intubation. The goals of preoxygenation are to completely saturate the oxygen-carrying capacity of all hemoglobin molecules (i.e., achieve an oxygen saturation of 100%), to denitrogenate the pulmonary functional residual capacity and maximize oxygen storage in the lungs, and to prolong the period of safe apnea between paralysis and successful tracheal intubation.

Oxygen desaturation to levels below 70% increases the risk of cardiac dysrhythmia, hemodynamic instability, hypoxic encephalopathy, cardiac arrest, and death. In patients for whom the room air oxygen saturation is 100% and who have no primary lung pathology with physiologic hemoglobin levels and low metabolic demands, the risk of critical desaturation is low following adequate preoxygenation. Conversely, any patient who does not meet the above criteria and is already hypoxemic (oxygen saturation < 90%) despite high-flow oxygen administration—that is, a significant number of patients requiring emergency airway management—is at very high risk for critical desaturation during efforts to secure a definitive airway.

Selecting the best method to adequately preoxygenate each patient will depend on the available equipment in a particular emergency department and on the patient's risk for desaturation. The goal, however, remains the same: to deliver the maximal fraction of inspired oxygen to the patient. The

standard ED nonrebreather mask without one-way valves covering all ports and set to a flow rate of 15 liters/minute (L/min) will deliver 60% to 70%  $\text{FiO}_2$  and will not provide complete denitrogenation. When the flow rate is increased to 30 to 60 L/min, however, these masks are capable of delivering  $\text{FiO}_2 > 90\%$ . For patients at low risk for desaturation, preoxygenation with a nonrebreather mask at high flow rates is recommended.

For patients at moderate or high risk for desaturation during intubation attempts or for whom the oxygen saturation is  $<93\%$  while receiving high-flow oxygen, preoxygenation with noninvasive positive pressure ventilation (NIPPV) is recommended. By increasing mean airway pressures, NIPPV allows these patients to overcome the shunt physiology that prevents adequate oxygenation despite maximal  $\text{FiO}_2$ . NIPPV in critically ill ICU patients has been shown to significantly affect patients' ability to improve their preintubation oxygen saturation and to maintain physiologic oxygen saturation levels during apnea. NIPPV may be achieved with the commonly available CPAP mask and ventilator circuit, or by assisting spontaneous ventilations with a standard bag-mask circuit and a positive end-expiratory pressure (PEEP) valve. However, one should avoid using bag-valve-mask devices for preoxygenation if not actively assisting spontaneous ventilations. Actively assisting ventilations would mean that a provider is squeezing the bag during inspiration and a tight mask seal is maintained. If not done properly, the patient will receive only atmospheric  $\text{FiO}_2$ .

In addition to selecting the appropriate method of preoxygenation, patient positioning is of paramount importance in achieving the goals of preoxygenation. Supine positioning prevents the patient from utilizing the full pulmonary vital capacity, resulting in atelectasis of the posterior aspects of the lungs and reduced oxygen storage in the pulmonary residual capacity. Studies of patient positioning have shown that patients preoxygenated in a 20-degree head-up position maintain their oxygen saturation for 20% to 30% longer during apnea compared to patients treated in the supine position. For patients unable to be placed in an upright position (e.g., those with potential spinal cord injury), similar benefit can be found by placing the patient in reverse Trendelenburg position.

Ideally, patients should receive preoxygenation for the maximum period of time prior to induction of apnea and airway manipulation, but this is not always possible in the emergency setting. Suggestions for empiric timing of the preoxygenation period include 3 minutes of a patient's normal respiratory pattern (tidal volume breathing) while receiving maximal  $\text{FiO}_2$ . To augment denitrogenation during this period, the patient should be instructed to fully exhale the expiratory reserve volume prior to beginning the 3-minute period



of tidal volume breathing. In patients able to comply, this 3-minute period may be reduced to 60 seconds by instructing the patient to take 8 maximal exhalations and maximal inhalations (vital capacity breathing) while receiving  $\text{FiO}_2$  of at least 90%.

The method selected for appropriate preoxygenation should be continued throughout the peri-intubation period from the administration of induction and paralytic agents until onset of complete muscle relaxation. In addition, recent literature has supported the use of apneic oxygenation throughout the peri-intubation period to delay desaturation. Despite the absence of active ventilation, alveolar oxygen diffuses into the blood while a lesser volume of carbon dioxide diffuses from the blood into the alveoli, creating a subatmospheric alveolar pressure gradient. This pressure gradient allows for spontaneous gas flow from the pharynx to the alveoli. When supplementary pharyngeal oxygen is supplied, this pressure gradient allows for continued and prolonged alveolar oxygenation—termed apneic oxygenation—despite the absence of ventilation. To accomplish apneic oxygenation, one places a nasal cannula on the awake patient underneath the nonbreather or NIPPV mask during preoxygenation. Upon induction of sedation and muscle relaxation, an assistant adjusts the nasal cannula oxygen regulator to a flow rate of 15 L/min and oxygen flow via the nasal cannula is continued throughout orotracheal intubation attempts. For patients who require NIPPV during preoxygenation, one may consider leaving the NIPPV device in place until the orotracheal intubation attempt is begun, as apneic oxygenation will not provide sufficient airway pressure to overcome the prevailing shunt physiology.

Though preoxygenation has been considered standard of care for over half a century, there are novel approaches to this standard practice. One such development is the use of ketamine to achieve dissociation while preserving airway reflexes in the patient unable to tolerate attempts at preoxygenation. This process, termed “delayed sequence intubation,” approaches preoxygenation similarly to any other emergency department procedure that requires sedation for success. Though this practice is not currently standard of care, it is an area of ongoing study.

## KEY POINTS

- Preoxygenation to extend the period of safe apnea is recommended for all emergency department intubations.
- Preoxygenation may be accomplished via nonbreather, NIPPV, or

bag-valve-mask devices, depending on individual patient parameters.

- Preoxygenation should occur with either a head elevated or reverse Trendelenburg position for maximal effect.
- Preoxygenation should occur for 3 minutes of tidal volume breathing or for 8 vital capacity breaths over 60 seconds.
- Preoxygenation should be augmented with apneic oxygenation during intubation attempts for all patients.

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# AIRWAY ADJUNCTS: KNOW YOUR BACKUP PLANS

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**NICHOLAS SAUBER, MD, RN-BSN, EMT-B**

Emergency airway management is one of the most high-risk and high-pressure procedures for the emergency physician. It comprises a large part of emergency medicine residency and continuing medical education training. Techniques and devices used in airway management have been widely studied. This has led to the development of a multitude of advanced airway devices to help ensure success. This chapter focuses on the backup airway devices and techniques including ventilation with bag valve mask and endotracheal intubation with direct laryngoscopy fail.

Before beginning, make sure to have the tools, supplies, and equipment necessary for airway management and have them close to the physician overseeing the airway. A video laryngoscopy cart or a size smaller tube can save a life, but not if it's neatly tucked away in the supply room during a difficult intubation. The popular SOAP ME mnemonic outlines the basic items you should have within reach at the head of the bed.

- **S:** Suction, on and working. This means tubing that's long enough with Yankauer attached and tucked under the patient's shoulder or mattress, within easy reach. Put it on the right side, in the same place every time, so you can grab it without taking your eyes off the airway while intubating.
- **O:** Oxygen, attached to devices and turned on. Multiple sources are ideal. A bag valve mask is standard, but consider a nonrebreather mask for preoxygenating a spontaneously breathing patient and a nasal cannula with high flow for passive (apneic) oxygenation during intubation attempts

- **A:** Airway equipment. This will differ depending on your facility and will be discussed in detail later, but the basics include laryngoscopes, endotracheal tubes (ETTs), stylets, supraglottic airways, video laryngoscopy, a cricothyrotomy kit, bougie, and a scalpel. Ensure that batteries and light sources are all working beforehand.
- **P:** Pharmaceuticals. Medications for induction, paralysis, intravenous fluids, and vasopressors if hypotension is expected. Also verbalize plan for post-intubation sedation.
- **M:** Monitors. Have the patient on a cardiac monitor. Turning on the audible pulse oximetry tone allows the intubator to be aware of the patient's oxygen saturation. Continuous end-tidal capnography will give visual confirmation of correct placement and help manage ventilator setting in the postintubation period. Blood pressure should be set to cycle at least every 5 minutes.
- **E:** Emergency equipment, including defibrillator and invasive airway equipment.

Once the first attempt at endotracheal intubation has been unsuccessful, the intubator has the option of attempting again, attempting another technique, or allowing another physician to rescue the airway. A 2005 study by Sagarin et al. showed that among emergency medicine residents, two-thirds who failed their first attempt at intubation had success on subsequent attempts. This should be balanced with the understanding that during intubation attempts, the patient is not being ventilated, and multiple or prolonged attempts may lead to fatal outcomes. Some organizations, such as the European Resuscitation Council, have recommended a 10-second time limit on all intubation attempts. A time limit should be established by the treatment team prior to intubation and should take into consideration the patient's condition and likelihood of desaturation (i.e., reserve). If direct laryngoscopy is deemed impossible, or if it is not quickly achieved, backup techniques should be employed with ventilation occurring between attempts. Devices will vary by institution, but the most common are described here briefly.

A gum-elastic bougie, or Eschmann stylet, is much longer, softer, and more flexible than a typical wire stylet and can offer an advantage when the vocal cords cannot be clearly visualized. The tip is angled at 30 degrees so that when it is inserted under direct visualization toward the glottis with the tip angled anteriorly, it enters the glottis opening and bumps or clicks along the tracheal rings. Its length allows an ETT to be threaded over the Bougie prior to insertion, or the ETT can be loaded by an assistant. This useful device should be within reach for every intubation.

Video laryngoscopy has become ubiquitous in emergency departments,

ranging from small hand-held devices with built-in LCD screens to larger devices with separate screens that can project images remotely. Broadly, intubation with video laryngoscopy involves 3 steps: exposing the larynx, positioning the tip of the ETT at the glottic opening, and advancing the ETT into the trachea. Each device has its own benefits, and there are too many to cover each in detail here. The main advantages of these devices, as a whole, is direct view of the larynx and glottis opening where line of sight is not possible through direct laryngoscopy, as well as minimal movement of the cervical spine. In addition, video laryngoscopy is a useful teaching tool as it allows the instructor to directly visualize the learner's technique. It is the responsibility of the emergency physician to become adept with the specific equipment at his or her own institution.

The final category of backup airway devices is the supraglottic airway. This category ranges from the emergency medical services (EMS) focused King laryngeal tube or Combi-tube to the laryngeal mask airway (LMA), more commonly used in the hospital setting. The most important advantage of the supraglottic device is that it is inserted blindly. Blind insertion eliminates the need for multiple laryngoscope blades and handles. Prehospital, military, and tactical medical providers utilize these devices as they are low tech and easily portable. In the hospital setting, where equipment is readily available, blind insertion is beneficial when the view of the vocal cords is obscured for various reasons. In this scenario, when endotracheal intubation and bag valve mask fail, inserting a supraglottic device can be a life-saving temporizing measure until a definitive airway can be placed.

When approaching the emergency airway, have a system to prepare your equipment and supplies the same every time to have the best chance of success. Become familiar with the equipment at your institution and practice with each adjunct regularly. Always plan for failure and have several backup plans at the ready.

## KEY POINTS

- Be methodical when setting up for emergency intubation and consider using a checklist approach.
- Be familiar with backup airway devices available at your institution. Know how to get them and when to use them.
- If available, the anesthesia team can provide airway adjuncts not available in the emergency department.

- A supraglottic device is extremely useful when endotracheal intubation and bag valve mask fail.
- Surgical airway is the final resort when all of the previously described techniques fail.

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# 4

## KNOW YOUR RSI MEDS

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**DANIEL SWEDIEN, MD**

Rapid sequence intubation (RSI) is a technique commonly used by physicians to manage emergency airways. This technique uses induction agents to bring about an unconscious state followed quickly by paralytic agents to aid in the rapid and safe intubation of an airway. It is critical to know a general approach to this and have a handle on the medications used in this process. This chapter focuses only on the RSI medications and bypasses other aspects in the process.

Every individual and institution will have different approaches to RSI, but it is important that physicians become comfortable with the steps and drugs they will use during the intubation process. Premedication, induction, and paralysis are the steps involving medications in RSI. Awake patients should be adequately sedated before being paralyzed. This can simply be done by having your induction agent pushed intravenously, followed immediately by your paralytic agent. It is also important to know your medications in this setting as your sedation can wear off while your patient is still paralyzed. This entire sequence is all time sensitive, so having an intimate knowledge of how your RSI medications will allow you to thrive in your approach to RSI.

Premedication is the use of agents such as fentanyl, lidocaine, and small doses of paralytics to blunt physiologic response to intubation. There is limited evidence to support this practice, as it may cause apnea before intubation. Recent evidence does suggest that patients unable to tolerate the preoxygenation period may benefit from small doses of ketamine (1 to 1.5 mg/kg) to improve oxygen saturations before intubation.

Sedation can be achieved by many routes, and currently, there are a variety of drugs used. Etomidate, ketamine, and propofol are three commonly used drugs for this, and each of them will have a distinct set of

advantages and drawbacks. One of the most commonly used medications etomidate (0.3 mg/kg/IV) has a quick onset of action and a short duration making it favorable as an induction agent. What also makes this drug one of the most commonly used drugs is that it is blood pressure neutral and lowers intracranial pressure. Without extensively listing them, there are drawbacks to etomidate. Recently, there has been some suggestion of increased adrenal dysfunction in patients with septic shock but no clear increase in mortality or resource utilization. Two other quick-acting induction medications include propofol (0.5 mg/kg/IV) and ketamine (1 mg/kg/IV). Propofol is a great anticonvulsant but is contraindicated in hypotensive patients. Ketamine is equally as effective as etomidate, preserves respiratory drive, but is contraindicated in hypertensive patients. Ketamine can be used as an alternative in septic shock patients. Remember when using sedative drugs to review your indications, contraindications, and onset of action/duration of treatment. This will allow you to adequately dose, redose if needed, and be able to switch between different sedatives as the clinical situation dictates.

Deciding on the best paralytic is similar to sedatives, in that there are advantages and drawbacks to available drugs. The most commonly used paralytics are depolarizing and nondepolarizing paralytics, with succinylcholine and rocuronium being the most common in each class respectively. Succinylcholine (1.5 mg/kg/IV) is the preferred paralytic as it is equally as effective as any nondepolarizing agent but has a rapid onset of action and shorter duration than any nondepolarizing agent. As with sedative medications, it is very important to switch paralytics based on clinical situation, and with succinylcholine, it is imperative to make a list of contraindications that would warrant use of a different paralytic. Common contraindications include recent burn, crush injury, denervation injuries or instances when a rise in serum potassium would not be tolerable, or if there was a concern for increased intracranial pressure or intra ocular pressure. Also, be aware that succinylcholine in emergency settings is likely to be underdosed, adding to difficulty in intubation. In these instances, rocuronium (1 mg/kg/IV) is often a suitable alternative. This drug is typically considered a second-line paralytic secondary to its longer duration of action. Recent evidence may also suggest that patients at high risk to desaturate may benefit from rocuronium as it may provide increased time before desaturation when compared to succinylcholine. What is important with paralytic drugs is to create either a mental or physical checklist that allows you to quickly assess if your medication is appropriate in your clinical situation or if alternates are needed.

Knowing your medications in RSI is important, as it will allow for increased success with intubations and fewer complications. The most



important take-home points are to know how to dose your medication and what clinical situation dictates deviating from the first-line agents. In the emergency situation, this can be difficult to assess rapidly, and thus, it is important to anticipate what situations should cause a physician to switch gears between his preferred RSI medications.

## KEY POINTS

- Induction and paralysis are the key steps involving medications in RSI.
- Etomidate, ketamine, and propofol are three commonly used drugs for sedation.
- Etomidate (0.3 mg/kg/IV) has a quick onset of action and a short duration and blood pressure neutral and lowers ICP. Ketamine (1 mg/kg) is contraindicated in hypertensive patients, is effective as etomidate, and can be used in sepsis. Propofol (0.5 mg/kg) is contraindicated in hypotensive patients and can be used as an anticonvulsant.
- Succinylcholine (1.5 mg/kg/IV) is a preferred paralytic, and as a depolarizing agent, it is equally as effective as any nondepolarizing agent, has a rapid onset of action and short duration, but is contraindicated in patients at risk for hyperkalemia, increased intracranial pressure, or increased intraocular pressure.
- Rocuronium (1 mg/kg/IV) can be a suitable alternative as a nondepolarizing paralytic agent.

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# **DID YOU MAXIMIZE YOUR LARYNGEAL VIEW?**

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**DEVIN M. KEEFE, MD**

When faced with imminent airway compromise, it is imperative that the emergency physician control as many variables as possible and maximize laryngeal view to ensure first-pass intubation success. With each repeat orotracheal intubation attempt, the incidence of adverse events increases significantly.

## **PREPARATION**

In preparation for RSI, all necessary airway adjuncts and backup devices should be at the ready. This includes, but is not limited to, a handle with tested light source, various sizes of Macintosh blades, straight (Miller) blades, a video laryngoscopy (VL) device, two sources of confirmed working suction, tested endotracheal tubes (ETs) of different sizes with stylettes in place, a bag-valve-mask, end-tidal CO<sub>2</sub> detector, stethoscope, naso-/oropharyngeal airways, 10-blade scalpel, bougie, and supraglottic rescue airway devices. Remember that the only failed airway is the one you do not anticipate. Preparation of backup measures (including a surgical airway) is essential.

## **POSITIONING**

Successful laryngoscopy begins with patient and operator positioning. The patient should be moved toward the head of the bed such that the glottic structures are no more than 18 inches from the laryngoscopist. The bed height should be adjusted so the intubator may stand up naturally while

introducing the laryngoscope blade with the left arm between 90 degree flexion and full extension at the elbow.

Extension of the patient's neck into ear-to-sternal notch alignment should be achieved. In the pediatric patient or morbidly obese, a neck roll or pillow may be needed to maintain this position. The patient's face will be roughly parallel to the ceiling with proper alignment. This position has been shown to maximize the distance between the palate and the tongue. Cervical extension beyond this will distort the anatomy and obscure the view.

After appropriate preoxygenation and RSI medications have been administered, the mouth is opened using a cross-scissor technique with the thumb and middle finger of the right hand. The incisors should be fully separated. Anterior digital pressure on the mandibular incisors by the thumb promotes anterior mandible sliding and provides an internal jaw-thrust effect maximizing space in the pharynx.

To achieve the best mechanical advantage, the laryngoscope handle should be gripped as low as possible and the elbow should be kept in close to the body. The Macintosh blade is introduced into the mouth by rotating the handle down and to the right. This minimizes handle contact with the patient's chest and allows the blade to drop into the oropharynx while avoiding teeth and soft tissue structures. The blade can now be rotated to the midline, sweeping the tongue gently to the left. The blade is advanced along the tongue to the base. It is at this point that the handle is distracted along its axis to move the tongue anteriorly. With the epiglottis in view at this point, the curved blade tip should be advanced to engage the vallecula. Proper blade tip seating in the vallecula allows the epiglottis to be lifted anteriorly exposing the glottic structures. Overinsertion promotes epiglottic closure while underinsertion will not transmit force necessary to lift the epiglottis via the hyoepiglottic ligament. Accordingly, small movements of the blade at the vallecula may yield large improvements in glottic view.

The glottic structures to be identified include the posterior arytenoids, vocal cords, and anterior commissure. The most common grading scale of laryngoscopic view is the Cormack-Lehane classification system with Grade 1 as full view of glottis and Grade 4 as neither glottis nor epiglottis in view.

Another, perhaps more intuitive, grading scale is the percentage of glottis opening (POGO) score. The POGO score represents the linear span from the interarytenoid notch to the anterior commissure of the cords. A 100% score is a full view.

Additional techniques such as bimanual laryngoscopy may play a role in augmenting a difficult view. Bimanual laryngoscopy using external laryngoscopy manipulation (ELM) is an airway technique that is performed

during laryngoscopy. The intubator reaches around the patient's head and manipulates the larynx with the right hand. Commonly, the thyroid cartilage is moved superiorly against the tip of the laryngoscope blade in the vallecula to help elevate the epiglottis. Once the desired view is obtained, the laryngoscopist asks an assistant to maintain that same pressure on the larynx so that the right hand can then be used to introduce the ET.

There is very little evidence to support cricoid pressure (CP) as an adjunct to laryngoscopy. The 2010 AHA guidelines do not recommend using CP during cardiac arrest. CP was not designed to improve laryngeal view, leads to complications, and should be generally avoided. Contraindications to CP include cricotracheal injury, active vomiting, unstable C-spine, poor laryngoscopic view, and LMA insertion.

Another commonly applied external airway manipulation technique is BURP (backward, upward, rightward pressure). This technique is similar to bimanual in that it involves ELM but is instead performed blindly by an assistant.

A 2006 study in fresh cadavers clearly showed bimanual laryngoscopy to be superior to the assistant-applied maneuvers. CP and BURP actually made glottic views worse about 1/3 of the time overall.

It should be noted that a straight tip blade such as the Miller requires a different technique. The straight tip blade is meant to directly lift the epiglottis to reveal the cords. While narrower and offering less tongue control, it is a useful approach when managing the floppy and relatively long epiglottis of the pediatric patient.

The advent of the VL has changed the way many emergency providers approach the airway and has led to much debate. The advantages of VL include the ability to intubate when neck mobility or oropharyngeal opening is limited, and allows for supervising physicians to see and assist with intubation, as well as a theoretical reduction of airway trauma. Critics of VL contend that blood or secretions may easily obscure the view and there is a likelihood for equipment failure and a potential for physicians to rely on the technology and lose DL skills. Emergency physicians should, of course, familiarize themselves with the VL devices at their institutions and choose the most appropriate tool based on the clinical situation.

## KEY POINTS

- Proper patient positioning and posture are essential.

- Stop and think about mechanics! Use finesse, not force.
- Ensure curved blade engages vallecula and adjust with small movements.
- Bimanual laryngoscopy improves view. CP is out. Know your VL devices.

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# **DON'T FEAR THE BLADE: SURGICAL AIRWAY**

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**ERNEST MAVUNGA, MD, MSc**

A cricothyrotomy is indicated when attempts to oxygenate have failed or when attempts to intubate have failed or are not plausible as is the case with facial trauma or distorted facial anatomy.

The greatest concern in performing cricothyrotomy is the delay in performing the procedure. This is sometimes due to repeated failed attempts at nonsurgical efforts to establish an airway (intubating laryngeal mask airway, fiberoptic scope, lighted stylet).

Unfortunately, there are numerous studies that have revealed that most patients were already in cardiac arrest or bradycardic before cricothyrotomy. The likely reason for the delay is discomfort with the procedure. As such, the failure linked to cricothyrotomy is a result of the delay to performing the procedure rather than the failure in the procedure itself. Equally important is the preparation for the procedure. That said, just as one sets up a backup airway with the nonsurgical adjuncts, a surgical alternative should be included. If possible, prior to an intubation, the sequence of events should be outlined before attempting to intubate. The sequence should include a review of the indications when a cricothyrotomy should be performed. By doing so, in cases where there is no time for preparation, having departmental clarity as when to move to a surgical airway and departmental recommendations in the steps for surgical airway management can be lifesaving.

## **CONTRAINDICATIONS**

Prior to cricothyrotomy, one should be familiar with the absolute as well as relative contraindications to performing a cricothyrotomy. In a life-

threatening airway emergency, there are few if any contraindications to cricothyrotomy. However, something to consider as a possible contraindication would be an obstruction distal enough to the cricoid membrane that a cricothyrotomy would not provide the required oxygenation or ventilation. Even in those cases, if the obstruction is mobile such as a food bolus, one could still perform a cricothyrotomy and push the bolus forward with the intent to right mainstem and then intubate the unobstructed side.

Relative contraindications to consider include prior neck surgery, obesity, neck pathology, prior radiation therapy to the area, coagulopathy, trauma/burns, hematoma, or any such features that distort anatomic landmarks. With these, one has to anticipate difficulties and so prepare accordingly and maybe consider consulting surgery, anesthesiology, or ENT early prior to the procedure.

## **PREPARATION**

As is the case with any procedure, success hinges on preparation. At the very least, prior to the procedure, one needs cuffed, nonfenestrated, No. 4 and No. 5 tracheostomy tubes and a number 11 scalpel. If available, include a tracheal hook, Trousseau dilator, 4 × 4 gauzes, suction, 2 small hemostats, antiseptic swabs, and surgical drape.

## **PROCEDURE**

Once the decision is made to perform a cricothyrotomy, start by identifying your landmarks and getting comfortable with positioning. The cricothyroid membrane is a dense fibroelastic sheet between the thyroid and cricoid cartilages with an average height and width of 10 mm (index finger width). Start by identifying the thyroid cartilage, which in males has a superior notch and is the largest laryngeal prominence. Whereas in females, the cricoid cartilage is the largest prominence, best identified by moving the palpating finger upward from the sternal notch. The cricothyroid membrane will then be the depression noted between the thyroid and cricoid cartilages. Once identified, sterilize the area. If possible, try to have a second pair of sterile hands available. If time permits, try to anesthetize the area locally. Thereafter, confirm your landmarks again, hold the trachea with your nondominant hand, and then make a 2- to 3-cm midline vertical incision through the skin. If the incision is not deep enough, one might need surgical clamps to spread the tissue in an effort to reach the cricothyroid membrane. Once identified, make a 1- to 2-cm transverse incision through the cricothyroid membrane. Afterward, insert your little finger into the incision



and through the cricoid membrane and feel the inside of the trachea; if you feel ridges, you're in the right place. With the finger within the opening, slide the tube straight down along the handle until it hits the rings in the back of the trachea, and advance until cuff is within the airway. Inflate the cuff; auscultate for breath sounds as well as use end title CO<sub>2</sub> for confirmation of airway placement. Given this is a shorter airway that might now be wet from the surrounding bleeding, extra caution should be exercised when securing airway.

If a hemostat and tracheal kit are available, then after horizontal incision is made, a hemostat can be used to open the airway. Thereafter, insert a tracheal hook into the opening, hooking the caudal end of the opening, and lift, allowing for passage of an appropriately sized cuffed endotracheal or tracheostomy tube (usually No. 5 or No. 6), directing the tube distally. Tube can then be secured after placement is confirmed.

## POST CRICOTHYROTOMY

After securing the airway, one needs to be weary of the complications of performing a cricothyrotomy and document appropriately. Commonly cited complications include aspiration, mediastinal emphysema, hemorrhage, creation of false passage into the tissue, esophageal/tracheal laceration, or vocal cord injury. The above complications are important to document so as to accommodate appropriate future management.

A cricothyrotomy can be a stressful situation, however, and can also serve as a great learning opportunity. For the stressful component of this procedure, a debriefing session would be helpful. During this session, discussing what went well and what didn't would be a good way to start. Thereafter, making sure everyone is heard and concerns are acknowledged is equally important. Some might consider cricothyrotomy to be excessive and unnecessary, and so acknowledging and encouraging discussions in this manner is important. Along the same lines, these cases can be the foundation for improvement of the procedure.

### KEY POINTS

- Have either an internal algorithm or documented department algorithm for when you will go to a surgical airway.
- Do not delay cricothyrotomy; a delay could result in a worse outcome.
- At the bare minimum, all you need is a No. 11 blade and cuffed,

nonfenestrated, No. 4 and No. 5 tracheostomy tubes, even a regular orotracheal tube size 7 (be weary of the length).

- Once one has entered the cricothyroid membrane, make sure to always have either a surgical device or your finger in the opening at all times until the tube is placed.
- Make room and time for a debriefing procedure; this can be a stressful experience for all involved.

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# **DO NOT RELY ON CLINICAL EXAMINATION ALONE FOR CONFIRMATION OF ENDOTRACHEAL TUBE PLACEMENT**

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Intubation is a critical procedure. The consequences of a malpositioned endotracheal tube can be disastrous, and therefore, confirmation of correct placement is essential. Direct visualization during endotracheal intubation (ETT) is the ideal technique to ensure proper placement but is not always possible and not always sufficient. Objective confirmatory findings are useful adjuncts that can help clinicians and other supporting team members confirm endotracheal positioning.

Clinically, equal bilateral breath sounds and an absence of audible gastric insufflation suggest a properly placed ETT. Additional reassuring signs after proper ETT include fogging of the tube with ventilation, equal chest rise bilaterally, and maintenance or improvement of SpO<sub>2</sub> levels on pulse oximetry. If auscultation reveals louder breath sounds on one side, the ETT has most likely been advanced too far and may be indicative of a right mainstem bronchus intubation due to the anatomical angle of the left mainstem bronchus. There may be other causes of unequal breath sounds such as pleural effusion, pneumothorax, hemothorax, obstruction, or consolidation that should be considered in patients with relevant risk factors.

There are several pitfalls when relying on clinical examination for

verification of proper tube placement. Gastric insufflation can still transmit breath sounds to the chest, providing a false reassurance. Fogging of the tube is neither sensitive nor specific for endotracheal positioning, but can be helpful when other confirmatory measures are used. Pulse oximetry should be monitored, but hypoxia can be a late finding of an improper intubation. A decrease in pulse oximetry may be a delayed finding of a malpositioned ETT and therefore is not a useful immediate adjunct for confirmation. Auscultating over the central chest may falsely mimic breath sounds with esophageal intubation, due to air passing through the esophagus. Additionally, equal breath sounds are appreciated in as many as 60% of right mainstem intubation.

There are multiple bedside tools available that should also be utilized to confirm endotracheal positioning. Qualitative colorimetric carbon dioxide (CO<sub>2</sub>) capnometers are commonly used and generally change from purple to yellow when exposed to CO<sub>2</sub> in the trachea, using pH-sensitive paper. False positives can occur with exposure to gastric CO<sub>2</sub>, but in this instance, the capnometer should change back to purple within six breaths. Thus, it is important to make sure color change is sustained when using a colorimetric capnometer. Continuous end-tidal CO<sub>2</sub> capnography works similarly, but provides a quantitative assessment of expiration of carbon dioxide and is a more reliable method of assessment. It has the additional benefit of providing information about quality of resuscitation efforts during a cardiac arrest; however, this is also a major limitation in confirming ETT placement as there may not be reliable CO<sub>2</sub> exchange with ventilation in a patient with severely limited perfusion.

Chest x-ray is another essential adjunct to ETT. It cannot reliably differentiate tracheal versus esophageal intubation, but rather should be used to determine proper depth of insertion, to rule out a mainstem intubation, and to identify other potential pulmonary concerns such as pneumothorax, hemothorax, pleural effusions, or consolidation. At the time of intubation, a depth of three times ETT size at the incisor teeth is appropriate (e.g., 21 cm for a 7.0 ETT). Radiographically, depth should be adjusted so the tip of the ETT is ~3 to 4 cm above the carina.

An esophageal detector device is another option available to confirm ETT placement. Although not commonly used in the emergency department, it is rapid and inexpensive and can be employed in the prehospital setting. This method involves syringe aspiration of the tube, with an adapter to connect the syringe to the ETT. When the ETT is correctly positioned, the clinician will encounter little or no resistance during aspiration. Conversely, when the ETT is in the esophagus, aspiration will lead to high resistance due

to collapse of the esophageal wall.

Ultrasonography provides two methods of ETT placement verification. The first involves using a linear probe at the cricothyroid membrane during the process of intubation. Increased shadowing of the esophagus should occur with esophageal intubation, causing a “double bubble” sign. The second method involves using a linear probe to verify bilateral lung sliding, similar to examination to rule out pneumothorax. Bilateral lung sliding would be consistent with a properly placed ETT. There are multiple methods of verifying correct endotracheal tube placement, with direct visualization and capnometry currently being the most reliable and widely used. The wide variety of techniques discussed in this chapter are essential, as use of multiple verification methods provides the highest degree of reassurance to the intubating clinician and the rest of the team that the endotracheal tube is where it needs to be.

## KEY POINTS

- The ideal way to confirm ETT placement is direct visualization of the tube passing through cords.
- The presence of bilateral breath sounds with equal chest rise and fogging of the tube can help to verify of ETT, but additional methods of confirmation should be obtained.
- Colorimetric and end-tidal CO<sub>2</sub> capnometry are invaluable adjuncts to proper ETT verification.
- Chest x-ray should be obtained post intubation, with the ETT tip ideally being 3 to 4 cm above the carina.
- Multiple methods of verification should be employed to confirm endotracheal positioning.

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# THE ART OF BAGGING

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**DEVIN M. KEEFE, MD**

The bag-valve mask (BVM) is a ubiquitous piece of resuscitation equipment used frequently in both prehospital and emergency department settings. Effective BVM ventilation is an essential skill. It can serve as a highly effective (albeit temporary) means of ventilation and oxygenation. Yet, the intricacies of proper BVM technique are often underappreciated and not formally taught, potentially resulting in inadequate or inappropriate ventilation and oxygenation.

## EQUIPMENT

The standard BVM is composed of a self-inflating, manually compressible bag attached to a nonrebreathing valve and face mask on one end and a reservoir that can be attached to an oxygen source on the other. Bags are made in various sizes for infant, children, and adult patients. In theory, with proper seal, the BVM can deliver 100% oxygen at a flow rate of 15 liters (L) per minute. Realistically, most systems deliver ~75% oxygen. For maximal oxygen delivery, the attached oxygen flow rate must exceed administered minute ventilation. An inlet valve allows room air to enter when reservoir volume is inadequate for bag filling.

Importantly, BVMs do not supply passive blow-by oxygen; the BVM is mostly meant to be used as a positive pressure ventilation device. Due to the nonrebreathing duck bill valve, oxygen is administered only when a sufficient pressure gradient is generated across the valve. In a spontaneously breathing patient (without bag compression), oxygen delivery is driven only by the patient's own negative inspiratory pressure. Without compression, BVM-administered  $\text{FiO}_2$  varies by device from 0.55 to 0.96 depending on the compliance of the manufacturer's valve. Accordingly, the BVM is less

effective in the spontaneously breathing patient and requires manual assist, that is, positive pressure compressions coordinated with the patient's own respirations. This is an important concept, as placing a BVM on a spontaneously breathing patient does not deliver a flow of oxygen in the same manner as a nonrebreather mask or nasal cannula. Commonly, unknowing providers place a static BVM on a patient in respiratory distress during the peri-intubation period, believing they are preoxygenating the patient. In actuality, this inappropriate use of the BVM can lead to poor oxygen delivery and rapid desaturation during rapid sequence intubation (RSI).

Whenever managing the airway, always ensure all necessary equipment is close at hand. This includes at a minimum, pulse oximeter, oxygen source, nasopharyngeal and oropharyngeal airways, tongue blade, water-based lubricant, and a Yankauer suction catheter with vacuum power source.

## **MASK SEAL**

Arguably, the most important element of good BVM ventilation is adequate mask seal. Masks come in a variety of sizes and should be chosen appropriately. Both under- and overinflation of the mask cushion may disrupt the seal. Several studies have investigated predictors of poor mask ventilation and compared mask seal techniques. Predictors of difficult BVM ventilation are intuitive and include obesity, Mallampati class III or IV status, advanced age, limited jaw protrusion, short thyromental distance, presence of facial hair, patients who are edentulous, and patients who snore.

In single operator BVM ventilation, a classic C-E technique is used with the thumb and index finger forming a "C" encircling and stabilizing the mask over the nose and mouth while the third, fourth, and fifth digits are in "E" formation along the mandible. Proper neck extension and mandibular protrusion pulling the jaw into the mask are vital to ensuring airway patency. Pushing the mask down onto the face promotes compression and difficult ventilation. Hart et al. prospectively studied one-handed versus two-handed mask seal on a training mannequin concluding that two-handed operation yielded higher tidal volumes and peak pressures indicating better seal. There are two generally accepted two-handed techniques for use when a second operator is available to compress the bag: a two-handed C-E technique, positioning both hands on either side of the mask as described above, and the V-E technique. In the V-E technique, the thumb and thenar eminence of both hands are positioned in parallel on either side of the mask connector while the second through fifth digits provide anterior jaw thrust on the mandible. There is no evidence supporting one technique over the other.



When discussing mask seal, the edentulous patient warrants special consideration. Repositioning the mask such that the caudal edge rests above the lower lip (i.e., moving the mask up the face so the bottom sinks into the mouth) may improve ventilation by opening the airway and allowing better jaw thrust. If dentures are available, they should be placed in the patient's mouth to optimize BVM technique.

## VENTILATION

A common acronym for proper BVM ventilation technique is JAWS:

- Jaw thrust
- Airway placement (oropharyngeal and/or nasopharyngeal)
- Work together
- Small and slow squeeze

Optimal manual compression of the BVM bag should deliver 6 to 7 cc/kg per breath over 1 to 2 seconds and not exceed a rate of 12 breaths per minute. Lower rates (6 to 8 breaths per minute) may be appropriate in patients with severe obstructive pulmonary disease to prevent air-trapping. Typical adult BVM bags are around 1.5 L, so keep in mind that manual compression of 1/3 of the bag will deliver a 500-mL tidal volume; additional compression will likely deliver unnecessarily large volumes. Paying close attention to rate and volume is critical; common issues with BVM ventilation are hyperventilation and inappropriate tidal volumes. Most BVMs have a pop-off valve that releases pressure in excess of 60 cm H<sub>2</sub>O. However, ideal pressures should be maintained below 30 cm H<sub>2</sub>O to eliminate barotrauma or gastric distention with subsequent aspiration. Many newer BVM setups also include a positive end expiratory pressure (PEEP) valve on the exhalation port that can be set to the desired pressure to mitigate end-expiratory alveolar collapse.

### KEY POINTS

- Ensure a high-flow oxygen source to maximize FiO<sub>2</sub>.
- Optimize mask seal with head extension, jaw thrust, and oral/nasal airway placement.
- Use two-operator BVM technique whenever possible.
- Mind your rate, volume, and pressures when bagging.
- Provide synchronized assist breaths when using BVM on a

spontaneously breathing patient.

- Insert dentures or adjust mask placement in edentulous patient.

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# **BP STILL LOW? POSTINTUBATION HYPOTENSION**

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**ALISON TRAVER, PA-C**

Management of critically ill patients includes establishing and maintaining a patent airway, which often leads to endotracheal intubation (ETI). In the emergency department, airway management is commonly performed via rapid sequence intubation (RSI) using fast-acting hypnotic and neuromuscular blocking agents.

Complications of intubation include hypoxemia, aspiration, and postintubation hypotension (PIH). PIH is loosely defined in the literature as any recorded systolic blood pressure < 90 mm Hg within 60 minutes of intubation. If the patient is already hypotensive and/or requiring vasopressors, it may not be safe to intubate, unless the airway is completely incompetent. By definition this is not PIH, but the approach to these patients is similar.

## **WHY DO WE CARE ABOUT THIS?**

Hypotension at any point is independently associated with higher in-hospital mortality and longer length of stay in the hospital and ICU. Hypotension after ETI occurs in about one-quarter of patients who undergo the procedure and may occur for a variety of reasons. Understanding the physiology behind PIH will help you prepare and manage the situation when it occurs.

### **1) Insufficient venous return**

- 1.1.** Hypovolemia often accompanies critical illness and predisposes a patient to poor perfusion.
- 2.2.** Sympathetic tone is abruptly removed with induction and

sedation, preventing the compensatory action of endogenous catecholamines that may have been maintaining blood pressure.

- 3.3. Administration of induction agents may directly cause smooth muscle relaxation and vasodilation, reducing systemic pressure.
- 4.4. Transition from spontaneous ventilation (negative pressure) to mechanical ventilation (positive pressure) raises pressure transmitted to the right atrium.

## **TAKE-HOME**

Higher right atrial pressure and lower systemic pressure mean reduced venous return.

### 2) Predisposing factors

- 2.1. Comorbidities: Chronic obstructive pulmonary disease, diabetes mellitus, chronic kidney disease, left ventricular dysfunction, obesity
- 2.2. Risk factors: Older age, sepsis, preload dependence, adrenergic states, tenuous hemodynamics preintubation, tachycardia, angiotensin-converting enzyme (ACE) inhibitor use
- 2.3. Shock Index (SI)  $\geq 0.8$

## **TAKE-HOME**

The processes that lead to intubation are also commonly risk factors for complications associated with the intubation.

### 3) New or evolving medical problems

- 3.1. AutoPEEP, pneumothorax
- 3.2. acute coronary syndrome (ACS), PE, cardiac tamponade, arrhythmia
- 3.3. Hemorrhage, hypovolemia

## **How Do We Address PIH Using This Information?**

Try to predict who may be at risk for developing PIH. Optimize the hemodynamic status of these patients and try to prevent PIH from even occurring. Be prepared for it regardless of mitigation strategies. Be able to identify the problem early and then rapidly intervene.

## PREDICT AND PREVENT

Factors that are most helpful in predicting which patients will become hypotensive after intubation include SI, comorbidities, and age. Calculate SI by dividing **heart rate/systolic blood pressure**. Determine comorbid conditions and age.

Assess volume status. Unless patient are floridly volume overloaded, give fluids empirically. Hang fluids in a pressure bag, which can be inflated if needed. Ensure adequate intravenous access—two IVs should be in place.

In high-risk patients, you may consider starting a vasopressor before intubation or having it mixed and available at the bedside. Depending on department policies, you may consider having vasopressors available to administer as small boluses (push dose pressors). Epinephrine and phenylephrine are good choices. Phenylephrine is probably better in the tachycardic patient, given that it is a pure alpha-adrenergic agonist.

For the procedure, choose the best medications for the clinical scenario and adjust the dose. All induction agents will cause hypotension in a compromised patient due to sympatholytic properties; therefore, dose adjustment becomes very more important in preventing complications.

## REDUCE THE DOSE OF INDUCTION AGENTS

Propofol should be reduced by 90%. Etomidate may be reduced by 50%, although the literature is limited about whether there is sufficient anesthesia at lower levels. Ketamine is beneficial in at-risk patients because it offers a sympathetic surge and has a fast onset. Aim to provide a dose in between those recommended for pain management and dissociation; 0.5 mg/kg is probably a good place to start. **Paralytics should be dosed higher** if hypotension is present or if cardiac output is diminished. For example, succinylcholine may be increased to 2 mg/kg and rocuronium to 1.6 mg/kg.

## IDENTIFY AND INTERVENE

Know what is happening with the patient at all times. Monitor blood pressure frequently. You may even consider an arterial line. If you identify PIH, be systematic in your interventions:

- 1) Open fluids and pump up pressure bag. Ensure adequate preload.
- 2) Listen for bilateral breath sounds. Feel for subcutaneous emphysema. Rule out pneumothorax; decompress the chest if necessary.

- 3) Feel pulses. Resuscitate as needed per ACLS guidelines.
- 4) Get an electrocardiogram (EKG). Rule out acute coronary syndrome and arrhythmias.
- 5) Perform bedside ultrasound of the chest. Assess cardiac activity. Rule out cardiac tamponade. McConnell sign may reveal ventricular strain and pulmonary embolism.
- 6) Disconnect the ventilator. Rule out breath stacking and autoPEEP.
- 7) Start a vasopressor (infusion or push dose).
- 8) Repeat labs. Rule out acid-base disorder.
- 9) Change ventilator settings. Make modifications based on lung-protective guidelines and the underlying process.
- 10) Place a nasogastric tube to reduce gastric distention, which may be increasing intrathoracic pressure.
- 11) Modify postintubation sedation. Start low, but make the patient comfortable. You can give vasopressors and pain/sedative medications simultaneously. Consider changing to an alternative agent if the medication (i.e., propofol) directly causes hypotension.

## KEY POINTS

- Induction agents are sympatholytic and will cause hypotension in all patients who are maintaining hemodynamics with endogenous catecholamines.
- **Predict** patients at risk for PIH with SI and a thorough evaluation of age and comorbidities.
- **Prevent** PIH by optimizing volume status, empirically starting vasopressors, and dose-adjusting induction agents.
- **Identify** PIH early by staying near the patient and monitoring vital signs frequently.
- **Intervene** on PIH rapidly and systematically.

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- Laryngoscope as a Murder Weapon—Hemodynamic Kills: <http://emcrit.org/podcasts/intubation-patient-shock/>
- The frequency and significance of PIH during emergency airway management.
- Predictors of the complication of postintubation hypotension during emergency airway management.
- Troubleshooting Mechanical Ventilation: <http://www.aic.cuhk.edu.hk/web8/Mech%20vent%20troubleshooting.htm>

# **FINDING THE SITE: SITE SELECTION AND MINIMIZING COMPLICATIONS FOR CENTRAL LINE PLACEMENT**

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**DILNAZ PANJWANI, MD, FACEP AND RICHARD PAUL,  
MD**

## **AVOID ARTERIAL INJURY**

Arterial puncture and hematoma formation are the most common complications of central line placement. Often, these complications are a result of subsequent dilation and insertion of the catheter into an artery and not a result of a puncture with the initial access needle. Therefore, it is critically important to ensure venous cannulation and confirm the guidewire is passed into the venous system. Two techniques, ultrasound guidance and pressure measurement, have been shown to decrease the rates of arterial catheter insertion.

### ***Ultrasound Guidance***

It has been widely demonstrated that 2D dynamic ultrasound imaging, in which the tip of the needle is visualized in real time entering the intended vein, significantly decreases rates of arterial puncture. Many reports have revealed that arterial placement of the catheter still occurs despite the use of ultrasound needle guidance. Speculated causes include movement of the needle into the artery after removing the ultrasound probe, mistaking the



shaft of the needle for the tip, and creating an arteriovenous tract prior to finding the needle tip in the venous lumen. In order to minimize arterial cannulation, ultrasound can be used to confirm guidewire placement in the vein prior to dilation of the vessel.

## ***Pressure Measurement***

A second technique to avoid inadvertent arterial placement is measurement of pressure in the needle. Studies have shown that almost 1% of arterial punctures were not recognized by color and pulsatile flow of blood from the needle. A large retrospective analysis of over 9,000 central line placements with mandatory use of pressure measurement resulted in zero arterial catheter insertions. There are several ways to conduct pressure measurement during central line placement.

The first method of pressure measurement is to attach sterile tubing to the needle or short plastic catheter and hold it vertically while watching for the rise of blood. Blood that continues to rise and overflow from the tubing indicates arterial pressure, whereas blood that ceases to rise or gradually starts to fall back toward the needle indicates venous placement. There are also commercially available sterile manometers that can be used to confirm venous pressure. It should be noted that either the sterile tubing or manometer can be attached to either a needle hub or the short plastic catheter included in the kit. It is recommended to use the catheter, as manipulation of the tubing while attached to the needle can result in movement of the needle tip, either into the artery or out of the vein altogether. This method may not be useful in very hypotensive patients, as their low arterial pressure may be mistaken for a venous pressure.

## **SUBCLAVIAN APPROACH**

Site selection is critical in avoiding complications and optimizing success rates. The subclavian approach is useful for patients with cervical collars or patients with severe orthopnea who must remain in a sitting position. However, the subclavian vein is not in a compressible site, which limits the ability to apply compression in response to an arterial puncture should one occur. Further, the clavicle can decrease the ability to visualize the vein with ultrasound. The subclavian vein can be cannulated from a supraclavicular or infraclavicular approach. Reviews have demonstrated that the supraclavicular approach is less likely to develop complications such as an iatrogenic pneumothorax and has higher success rates than does the infraclavicular approach.

## INTERNAL JUGULAR APPROACH

The internal jugular approach allows superior ultrasound visualization compared to the other sites, both in locating the target vein and in demarcating adjacent arteries. Further, should arterial puncture be a complication, this site allows easy compression and visualization of any expanding hematoma. However, access to the internal jugular may be difficult in situations of chest compressions, complicated airway management, or trauma patients with cervical collars or neck injuries.

## FEMORAL APPROACH

The femoral approach is useful in a patient undergoing chest compressions, since the insertion site is located away from the moving chest wall. In addition, there is no risk of iatrogenic pneumothorax and the artery is in a compressible site. However, the long-term risk of catheter-associated deep venous thrombosis is significantly higher in femoral lines, and the rate of catheter-associated blood infections may also be higher in femoral lines, although the data surrounding this issue have provided mixed results.

### KEY POINTS

- Maximize success rate of venous cannulation by using real-time 2D ultrasound to visualize needle cannulation of desired vein.
- Use ultrasound to confirm wire placement throughout as much length of vein as possible prior to dilation and catheter insertion to avoid dilation of an artery.
- Use pressure testing with sterile tubing or digital manometry to confirm venous placement, but not in extremely hypotensive patients.
- Use the supraclavicular approach to subclavian lines to minimize risk of iatrogenic pneumothorax.
- Consider patient anatomy, clinical condition, and site-specific risks to select most appropriate approach for central line insertion in order to minimize short-term and long-term complications.

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## MANAGING CARDIAC ARREST

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Cardiac arrest (CA) can be defined as the acute loss of heart function, either instantaneously or following a range of symptoms, ultimately leading to an arrest of circulation.

Clinically, the three classic characterizing features of CA are pulselessness, unresponsiveness, and abnormal breathing—either agonal or absent. The estimated annual incidence of out-of-hospital CA ranges anywhere from 250,000 to 420,000 and is still one of most common causes of death in the United States. There is a multitude of underlying etiologies with ischemic heart disease being the most common. There are four main conduction rhythms of CA. The first two are ventricular fibrillation and pulseless ventricular tachycardia (VF/pVT)—these are shockable with a more favorable outcome. The other two are nonshockable and they include pulseless electrical activity (PEA) and asystole. Even in those patients managed in the prehospital setting by emergency medical service (EMS) personnel, the rate of survival to hospital discharge with meaningful neurologic outcome is roughly 8%. In some settings, however, survival rates have been shown to approach nearly 50% suggesting that there is still significant room for improvement in CA care. This disparity seems to lie not in what new or advanced technologies are being used, but rather, in how quickly and effectively the basic CA efforts are being performed.

Whether occurring at home, in the public setting, or in the hospital, prompt recognition of CA is key. Early activation of emergency response systems, initiation of high-quality cardiopulmonary resuscitation (CPR), and expedient use of automated external defibrillators (AEDs) are absolutely paramount. Swift deliverance of high-quality CPR can prevent progression of shockable rhythms to nonshockable rhythms and increase successful defibrillation rates, chances for survival, and neurologic integrity.

Unfortunately, less than half of patients with out-of-hospital CA will receive bystander CPR, and AEDs are used <10% of the time. Even with recent technologic advances, these basic components, especially early defibrillation and high-quality CPR, remain the mainstay of CA resuscitation.

In most cases, delivering a shock comes in a standardized, device-dependent fashion with the push of a button; the same cannot be said about CPR. There is much more to high-quality CPR than simply pushing on a patient's chest. This is critical because even though high-quality CPR is one of the few interventions that has been shown to improve CA outcomes, it is still performed ineffectively in many cases.

The most frequent deficits in high-quality CPR are inadequate rate and depth of chest compressions. With hands positioned on the lower half of the patient's sternum, compressions should be performed at a rate of 100 to 120 per minute at a minimum depth of 2 inches (5 cm) and no >2.5 inches (6 cm). Compressors should allow for full chest wall recoil in between compressions and avoid leaning on the chest.

Appropriate ventilation rates are also important. A single rescuator should follow a 30:2 compressions-to-rescue breath ratio. When additional personnel are available, one breath every 6 seconds (10 per minute) should be given.

Any pause in chest compressions should be limited to no longer than 10 seconds. This means that at no point in a CA resuscitation, for example, for rescue breaths, pulse checks, rhythm analysis, charging for defibrillation, etc., should compressions be held for longer than this. Compressions should be resumed immediately after shock administration and should constitute at least 60% of the total resuscitation time.

When high-quality CPR with minimal disruptions is effectively provided, observational studies have demonstrated more successful defibrillation rates, return of spontaneous circulation (ROSC), and survival to hospital discharge. There are additional therapies and other adjuncts that can be considered for CA care as well. Nevertheless, effective high-quality CPR and early defibrillation continue to remain two of the most beneficial and critical interventions.

In the emergency department or other appropriately equipped environments, more advanced treatments may be considered. Airway management adjuncts, physiologic monitoring, and ultrasonography are just a few of these. Medications in CA also have a role but will be discussed elsewhere. Use of these therapies should never take precedence over, nor cause prolonged breaks in, high-quality CPR.

Decision to place advanced airways, for example, an endotracheal tube (ETT), versus use of bag-mask ventilation is frequently encountered in CA. As of 2015, no studies had demonstrated definitive evidence to advise routine placement of advanced airways according to the AHA. Effective bag-mask ventilation, albeit less invasive, still requires considerable skill; therefore, advanced airway placement may still be considered depending on the expertise of the provider. If an ETT is placed, continuous waveform capnography, or end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>) monitoring, is the preferred, most reliable modality for confirmation and ongoing assessment of correct placement.

Waveform capnography is also one of several parameters that may be reasonable to utilize during CA care for ongoing physiologic monitoring. These can provide real-time feedback about the patient's condition and aid in assessing both CPR quality and in early detection of ROSC. At the time of this publication, no studies had definitively demonstrated improved survival or neurologic outcomes by using these, however. The 2015 Advanced Cardiovascular Life Support (ACLS) guidelines still do include cutoff values for ETCO<sub>2</sub> (<10 mm Hg) and diastolic relaxation phase on arterial waveform (<20 mm Hg) as values at which to encourage better CPR. They also note abrupt increase in ETCO<sub>2</sub> measurement over 40 mm Hg or spontaneous arterial pressure waves as indicative of ROSC.

Lastly, ultrasonography is slowly being incorporated into CA management. When an experienced ultrasonographer is present, this modality may be considered for both confirmation of ETT placement as well as evaluating for reversible causes of CA, for example, tamponade, pneumothorax, and hypovolemia in the presence of a PEA CA. Overall, data are still limited at this time to show any long-term benefits.

In general, CA resuscitations should always have one designated leader coordinating all efforts, paying close attention to ensuring proper administration of high-quality CPR. Also, family members should be allowed to be present if desired. Finally, a debriefing period after completion of the resuscitation should be considered to assess overall performance and recognize areas for future improvement.

## KEY POINTS

- Early recognition of CA with prompt implementation of high-quality CPR is critical and can improve survival outcomes.

- High-quality CPR includes ensuring adequate rate and depth of chest compressions, allowing for chest wall recoil, avoiding excessive ventilation, and limiting compression pauses to no more than 10 seconds with a minimum total time of chest compressions during a resuscitation of 60%.
- Early defibrillation for ventricular fibrillation and pulseless ventricular tachycardia improves survival outcomes and should not be delayed.
- Advanced and adjunctive therapies in CA may be reasonable to use in the appropriate settings as long as high-quality CPR is not interrupted. There are still too limited data to make any definitive recommendations for the routine use of these technologies.
- There should be one appointed leader in charge of resuscitation efforts, families should be allowed to be present if so desired, and there should be a short debriefing session following the resuscitation; these are important general points to consider in CA care.

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# **MEDICATIONS IN CARDIAC ARREST: TIME FOR A REQUIEM?**

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**BACHAR HAMADE, MD, MSc**

In 1968, Redding and Pearson showed that resuscitation of dogs in cardiac arrest due to ventricular fibrillation (VF) was more successful while using epinephrine; since then, the American Heart Association (AHA) has recommended the use of many drugs in its advanced cardiovascular life support (ACLS) and emergency cardiovascular care (ECC) algorithms. These drugs, however, have not withstood the test of time. Due to a lack of evidence showing any benefit, many of them have been omitted as the algorithms have been updated. Procainamide and buffers that were recommended in the 2000 ACLS guidelines were removed from the 2005 guidelines, and in the most recent ACLS guidelines, the use of atropine and lidocaine is no longer routinely recommended for pulseless rhythms. There are however medications, including some of the abovementioned, that are still recommended in special situations. The current medications recommended by the AHA for cardiac arrest will be discussed below.

## **EPINEPHRINE**

Epinephrine has been persistently recommended in the ACLS guidelines, despite the weak evidence supporting its use. In 2014, a large systematic review of the literature was able to identify only one randomized controlled trial (RCT) comparing epinephrine to placebo that showed higher rates of return of spontaneous circulation (ROSC) and survival to admission after out-of-hospital cardiac arrest, in favor of epinephrine. However, the trial showed no difference in long-term neurologic outcome and survival to discharge. Currently, the AHA still recommends administering a 1-mg dose

of epinephrine (1:10,000 concentration) via the intravenous (IV) or intraosseous (IO) route, or 2 to 2.5 mg endotracheally, every 3 to 5 minutes during cardiac arrest.

## **VASOPRESSIN**

In 2005, a meta-analysis of three RCTs that compared vasopressin with epinephrine as a first-line vasopressor in cardiac arrest did not show any difference in ROSC, survival to discharge, or neurologic outcome. In 2010, the AHA recommended that 40 units of vasopressin, either IV or IO, may replace the first or second dose of epinephrine. This has very recently changed in the new 2015 updates; due to its equivalence to epinephrine, the AHA has simplified its algorithms and no longer recommends the use of vasopressin.

## **AMIODARONE**

Amiodarone is a class 1, 2, and 4 antiarrhythmic, and its use is currently recommended for refractory shockable rhythms, namely, ventricular tachycardia (VT) and VF. A systematic review in 2013 showed no benefit of amiodarone compared to placebo with respect to survival to hospital discharge, but showed higher rates of ROSC and survival to admission. The 2010 ACLS guidelines recommend an initial dose of 300 mg amiodarone IV/IO followed by one dose of 150 mg for refractory ventricular fibrillation or pulseless VT.

## **LIDOCAINE**

In the 2010 ACLS guidelines, the AHA removed the use of lidocaine as a standard antiarrhythmic for shockable rhythms that were refractory to CPR and defibrillation. However, the guidelines still recommended the consideration of lidocaine if amiodarone was unavailable. Most recently in the 2015 AHA updates, the initiation or continuation of lidocaine may be considered immediately after ROSC, if the arrest was due to either VT or VF.

## **MAGNESIUM SULFATE**

Magnesium sulfate may be used for special situations in cardiac arrest. For torsades de pointes (polymorphic VT associated with prolonged QT interval), magnesium sulfate can be administered as an IV/IO bolus at a dose of 1 to 2

g diluted in 10 mL D5W.

## **SODIUM BICARBONATE**

In previous ACLS guidelines, the routine administration of sodium bicarbonate during cardiac arrest was recommended. However, this recommendation has long been removed for multiple reasons. By reducing systemic vascular resistance, sodium bicarbonate can compromise cerebral perfusion pressure. It may also create extracellular alkalosis, shifting the oxyhemoglobin saturation curve to the left, thereby inhibiting oxygen release to the tissues. Additionally, by producing excess carbon dioxide, it can paradoxically contribute to intracellular acidosis. In certain special situations, however, sodium bicarbonate does offer benefit. For example, in patients who have end-stage renal disease with preexisting metabolic acidosis or suspected hyperkalemia, or in tricyclic antidepressant (TCA) overdose, sodium bicarbonate may be beneficial by increasing serum pH and increasing sodium levels. In these cases, sodium bicarbonate (8.4% solution, 1 mEq/mL) can be administered in boluses of 1 mL/kg.

In conclusion, while the evidence behind the use of various medications in cardiac arrest is very weak, many of these drugs are still recommended to be used per the AHA guidelines. The clinician has to be vigilant in considering the potential benefit versus harm in administering these drugs during resuscitation.

### **KEY POINTS**

- Epinephrine is still the most widely used drug in cardiac arrest, regardless of presenting rhythm.
- Vasopressin is no longer recommended due to its equivalent nature to epinephrine.
- Amiodarone may be used in cardiac arrest with VT or VF refractory to defibrillation.
- Magnesium is recommended for polymorphic VT, also known as torsades de pointes.
- Sodium bicarbonate is recommended for severe acidosis, hyperkalemia, and TCA overdose.

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# **WHAT ARE YOUR VENT SETTINGS, BUD?**

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In the emergency department (ED), a lot of emphasis is placed on endotracheal tube (ET) placement, and as important as tube placement is, perhaps the real skill lies in adjusting those ventilator dials once the tube is placed. Patients are spending longer times in the ED than ever before and because ventilators are not benign devices, vent settings need to be optimized by ED providers. With so many different vent modes and settings, the question is where to start. The answer is at the beginning: by choosing the mode.

Assist control (AC) is the recommended mode in the ED for most patients. AC is advantageous not only because it is versatile mode that can be used for a myriad of indications including airway protection hypoxia, COPD, asthma, and metabolic derangements but also because the mode emergency medicine providers are most comfortable with. AC is a full support mode that delivers a set tidal volume ( $V_t$ ) at a set rate and for any breaths the patient triggers over the set rate, the entire  $V_t$  is given. For the sake of simplicity, all settings will be discussed with the assumption that the mode chosen is AC. Once the vent is on AC, there are four additional settings to dial in: tidal volume, respiratory rate, fraction of inspired oxygen, and positive end-expiratory pressure.

## **TIDAL VOLUME ( $V_T$ )**

$V_t$  is the volume of air the vent will deliver through the ET tube. For patients with normal lungs and in those with ARDS, using lower tidal volumes has

shown to decrease the development of lung injury as well as decrease mortality. In patients with normal lungs, a  $V_t$  of 6 to 8 mL/kg of *ideal* body weight is generally sufficient. For patients with asthma, COPD, or ARDS, who are at increased risk for ventilator-associated lung injury (VALI), tidal volumes of 6 mL/kg should be used in order to adequately protect the lungs. Ideal body weight should be determined using the formula  $50 + 2.3 [\text{height (inches)} - 60]$  for males and  $45 + 2.3 [\text{height (inches)} - 60]$  for females.

## RESPIRATORY RATE

In patients without obstructive pathology (asthma, COPD) or hypercapnia, start at a rate of 16 to 18. Patients with obstructive pathology need rates of 10 to 12 in order to prevent VALI, while patients with hypercapnia may require higher respiratory rates in order to help the patient blow off excess  $\text{CO}_2$ . In these acidotic patients, start at a rate of 20, and repeat blood gases frequently to titrate the rate. While it is tempting to try to increase the respiratory rate in order to drive down the  $\text{PaCO}_2$  to make the blood gas values look near normal, higher  $\text{PaCO}_2$  levels are acceptable and often necessary in these vented patients. This practice called permissive hypercapnia remains tolerable as long as the pH remains  $>7.2$ . A  $\text{pH} < 7.2$  is thought to be associated with decreased tissue perfusion and oxygenation and should therefore be avoided.

## FRACTION OF INSPIRED OXYGEN ( $\text{FiO}_2$ )

$\text{FiO}_2$ , the percent of oxygen in the measured air, should be started at 100 postintubation in order to ensure that the patient becomes stabilized quickly. The 100% oxygen provides protection against hypoxia that may have occurred before or in the process of placing the tube, but oxygen toxicity has proven to be deleterious to patient outcomes and should be avoided as well. While not completely understood, it is theorized that overoxygenation leads to the formation of oxygen free radicals that leads to inflammation and possible cell death. For these reasons, once the tube is secured, drop the  $\text{FiO}_2$  to 40% immediately after intubation in patients intubated for airway protection or for increased metabolic requirements. In patients intubated for hypoxia or obstructive pathology dropping, the  $\text{FiO}_2$  should be done with more care and in small increments but should be done nonetheless to avoid oxygen toxicity. Decrease the  $\text{FiO}_2$  after drawing the first blood gas and confirming sufficient oxygenation. Continue to maintain oxygen saturation

between 88% and 92% for COPD patients and between 90% and 95% for all other patients. It is not advisable to keep patients at a saturation of 100% because a saturation of 100% does not directly correlate with PaO<sub>2</sub>. Instead use the blood gas to determine oxygenation status and prevent overoxygenation.

## **POSITIVE END-EXPIRATORY PRESSURE (PEEP)**

PEEP is the pressure above atmospheric pressure that exists in the lungs at the end of expiration, and in the interest of keeping it simple, consider starting with a PEEP of 5 cm H<sub>2</sub>O in all patients. A PEEP of 5 should not cause any harm to your patient regardless of their reason for intubation and is useful in reducing atelectasis by preventing a decreased functional residual capacity in normal lungs. Consider going up on the PEEP if the patient is still hypoxic despite a FiO<sub>2</sub> of 100%. Increase PEEP by 3 to 5 cm H<sub>2</sub>O at a time.

Once the vent has been set to AC and V<sub>t</sub>, RR, FiO<sub>2</sub>, and PEEP are programmed, there are a few advanced maneuvers that can maximize ventilation: changing the inspiratory to expiratory ratio (I/E ratio), checking the plateau pressures (PP), and proning the ARDS patient when appropriate.

## **INSPIRATORY TO EXPIRATORY RATIO**

The I/E ratio can be adjusted to help decrease hypercapnia. The normal I/E ratio preset on the vent is 1:2, and this is suitable for most patients. In patients with obstructive airway disease, consider changing the I/E ratio to 1:4 or 1:5. Adjusting the ratio allows for less air-trapping secondary to breath stacking and incomplete expiration (auto PEEP). Both air-trapping and auto PEEP are detrimental in that they impede venous return and lead to decreased expiration. In order to adjust the I/E ratio, change the inspiratory flow rate (V<sub>i</sub>). The IFR, the rate at which a breath is delivered, is automatically set to 60 L/min, but can be increased to 100 L/min to allow tidal volumes to administer more quickly and thus allow for prolonged expiration.

## **PLATEAU PRESSURE**

The PP, the pressure in the small airways and alveoli, can be checked by pressing the inspiratory pause button. Ask the respiratory therapist for assistance about how to execute if needed. If the PP is >30, the patient is at risk for barotrauma, and the tidal volume needs to be lowered to prevent

alveolar damage. Decrease  $V_t$  by 0.05 to 1 mL/kg and recheck.

## ***Proning***

Placing a patient with ARDS on their stomach has been shown to increase oxygenation when compared to supine positioning. In addition to increasing oxygenation, the mortality rate has shown to be improved in prone patients in a head to head trial of prone versus supine ARDS patients.

Once the initial vent settings are programmed and any fine tuning completed, there are a few different ways to assess whether or not ventilation goals are being met: blood gas, continuous capnography, and the all-important visual evaluation of the patient's comfort. When checking blood gas, wait 20 to 30 minutes after setting adjustment to allow for blood gas stabilization and an accurate assessment of ventilation status. Continuous capnography can be useful as well because it reflects the lower limits of the patient's actual  $\text{PaCO}_2$ . Although it does not accurately correlate with  $\text{PaCO}_2$ , it can be a starting point. For example, if the patient's  $\text{CO}_2$  continues to increase after intubation, capnography illustrates that the patient is likely becoming more acidotic. Knowing this, adjustments can be made without waiting for the results of the gas. Lastly, simply observing the patient can give you an indication of how successful your settings are. If the patient looks uncomfortable and is fighting the vent, there is one of two problems: the patient is not sedated properly or the discomfort is secondary to air hunger. If sedation is sufficient, consider making vent adjustments sooner rather than later.

## **KEY POINTS**

- AC is the recommended mode in the ED for most patients.
- Tidal volume ( $V_t$ ) of 6 to 8 mL/kg of *ideal* body weight is recommended for patients with normal lungs.
- A respiratory rate of 16 to 18 breaths per minute is good place to start for a patient with normal lungs, whereas in patients with hypercarbia, consider 20 bpm, and in patients with obstructive pathology, consider 10 to 12 bpm.
- In most patients, start with a PEEP of 5 cm  $\text{H}_2\text{O}$ .
- Transition down from 100%  $\text{FiO}_2$  to 40%  $\text{FiO}_2$  as soon as possible.



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# **AFTER THE CARDIAC ARREST: POSTARREST CARE**

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**CHIDUBEM ILOABACHIE, MD**

Achieving return of spontaneous circulation (ROSC) after a cardiac arrest is one of the most satisfying and encouraging experiences in emergency medicine. It can be easy to forget that work yet remains to maximize the patient's likelihood of neurologic recovery. Though research in the field is limited, there is literature to support objectives that every provider should consider when caring for a patient who has been resuscitated after an arrest.

## **MAXIMIZE PERFUSION**

The first objective is to optimize cardiopulmonary function and, by consequence, end organ perfusion. Hypotension, typically defined as a mean arterial pressure (MAP) < 65 mm Hg or systolic blood pressure < 90 mm Hg, should be avoided. Starting with a crystalloid bolus is reasonable in all cases, although those who have arrested as a result of hemorrhage are likely to benefit more from blood products. Vasopressors and inotropes should be added when hypotension persists. This scenario is more likely when impaired cardiac function (i.e., myocardial infarction, severe sepsis) is the likely primary etiology of the arrest.

Similarly, hypoxia should be avoided. If there is a concern for ongoing hypoxia and only a supraglottic ventilation strategy was employed, consider conversion to a definitive airway using an endotracheal tube. Place the patient on a ventilator with a full respiratory support, particularly if the patient is not following commands. With respect to oxygenation, start with a fraction of inspired oxygen (FiO<sub>2</sub>) of 100% and peak end-expiratory pressure (PEEP) of 5 mm Hg. Titrate to keep the oxygen saturation above 94% and

perhaps not >98%. The upper parameter has no basis in the literature and only serves as a reminder of the danger of hyperoxia. The associated free-radical formation may worsen neurologic outcomes.

Either from chest compressions or as a result of the process that caused the arrest—or both—the patient may develop acute lung injury ( $\text{PaO}_2/\text{FiO}_2 < 300$ ) or acute respiratory distress syndrome (ARDS,  $\text{PaO}_2/\text{FiO}_2 < 200$ ). Therefore, consider lung protective ventilation strategies. Specifically, the goal tidal volume should be 6 mL/kg of predicted body weight. Caution should also be taken against hyperventilation as it may decrease cerebral perfusion. In healthy brain tissue, one can expect a decrease in cerebral perfusion of 2% to 4% for every mm Hg decrease of  $\text{PaCO}_2$ . Ventilation should be titrated to a goal  $\text{PaCO}_2$  of 40 to 45 mm Hg. It is important to remember that tachypnea may also be mediated by anxiety, pain, and/or agitation, which are all likely in the postresuscitated period. Consequently, analgesics and sedatives should be considered for patients who are not completely comatose.

## **DETERMINE THE ETIOLOGY**

The second objective is to ascertain and treat the cause of the arrest, if it can be identified. This will typically involve a broad range of bedside, laboratory, and radiographic tests. Obtain an electrocardiogram (EKG) as soon as the patient is stabilized to look for signs of ischemic heart disease. Evidence of an acute coronary syndrome (ACS) should prompt consideration for immediate treatment, including percutaneous intervention. Fibrinolysis or embolectomy can be performed when pulmonary embolism is the suspected etiology of the arrest. The remainder of the “H’s and T’s” should be considered and addressed based on clinical scenario. They include hypoxia, hypoglycemia, hyper-/hypokalemia, hydrogen ion (meaning acidosis of any etiology), hypovolemia, hypothermia, tamponade (pericardial), toxins, trauma, tension pneumothorax, and thrombosis, as discussed.

When ascertaining the cause of the arrest, consider whether an abnormal finding reflects the cause of the arrest or simply its sequela. For instance, severe acidosis—demonstrated by a high lactic acid, low pH, and low bicarbonate—may simply reflect the prolonged state of hypoperfusion rather than the etiology of the arrest. Therefore, treatment with a bicarbonate drip or with hyperventilation would not be appropriate; optimizing cardiopulmonary function and thus restoring perfusion would be expected to provide more benefit.

# OPTIMIZE NEUROLOGIC RECOVERY

The third objective is to maximize the environment for neurologic recovery. The literature behind this is not as definitive, but there are certainly common practices that ought to be considered. Hypothermia is perhaps the most discussed therapy. While the literature is equivocal on its benefit with respect to normothermia, it is clearly favorable when compared to hyperthermia. Hyperthermia, which may be related to ambient temperature or release of inflammatory cytokines, has a deleterious effect on the brain and should be actively avoided. Cold crystalloid infusion and external cooling are typically effective, and more invasive measures are seldom required. A reasonable target temperature is 36°C as it is just as effective as 32°C and has fewer associated complications (i.e., infection, coagulopathy, and cold diuresis).

Hyperglycemia has also been found to be associated with poorer outcomes and worse neurologic recovery. Treating it, however, needs to be balanced against the more precipitous threat of hypoglycemia. As such, moderate glycemic control is typically preferred, usually defined as a glucose level between 144 and 180 mg/dL.

Routine administration of neuroprotective pharmacologics has not demonstrated clear benefit and should be avoided. This includes but is not limited to coenzyme Q10, corticosteroids, thiopental, and magnesium sulfate. The one possible exception is antiepileptic therapy as the harm of sustained epileptiform activity on brain tissue is clear. However, without an electroencephalogram, diagnosis of seizure is not easily made.

The ultimate disposition of the resuscitated patient postarrest is the intensive care unit. There, intensivists along with various other subspecialists can optimally manage the patient, hopefully affording him or her the best possible outcome. The patient's family or other surrogate decision-makers should be involved early and often. As patients obtain ROSC and stay in the emergency department for at least a short period of time, it is the responsibility of the emergency medicine clinician to initiate the best possible postarrest care early on.

## KEY POINTS

- Perfusion should be maximized by focusing on cardiac output and tissue oxygenation.
- After ROSC is achieved, search for the underlying etiology of the cardiac arrest.

- Various approaches exist to maximize the most meaningful outcome, neurologic recovery.
- Care should be coordinated among various subspecialists as well as the patient's family or other surrogate decision-makers.

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# COOLING, How Low Do You Go? THERAPEUTIC HYPOTHERMIA IN THE POSTARREST PATIENT

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**BACHAR HAMADE, MD, MSc**

In 2005, the American Heart Association (AHA) guidelines for cardiopulmonary resuscitation and emergency cardiovascular care introduced therapeutic hypothermia to the post-return of spontaneous circulation (ROSC) algorithm in comatose patients. This was a major difference from the 2000 advanced cardiovascular life support (ACLS) guidelines, which clearly stated that hypothermia should not be induced after resuscitation from cardiac arrest (though they did acknowledge a potential benefit in patients who spontaneously developed mild hypothermia  $> 33^{\circ}\text{C}$ ).

The changes were prompted by two trials published in the year 2002 that clearly showed favorable neurologic outcomes in patients cooled to hypothermic levels, compared with normothermic patients, after ROSC.

In the first study, Bernard et al. randomized 77 eligible patients (comatose survivors of out-of-hospital cardiac arrest) to treatment with hypothermia versus normothermia. Target core temperatures were  $33^{\circ}\text{C}$  and  $37^{\circ}\text{C}$ , respectively. The patients randomized to the hypothermia group were brought to goal temperature within 2 hours of ROSC, were maintained at target core temperature for 12 hours, and were then actively rewarmed to normothermia. At hospital discharge, 49% of the patients in the hypothermia group were considered to have a good neurologic outcome (discharged to home or rehabilitation facility), compared to only 26% in the normothermia group.

In the second study, the authors (the Hypothermia after Cardiac Arrest

[HACA] study group) randomized patients who had been resuscitated after cardiac arrest due to an initial rhythm of ventricular fibrillation (VF) to undergo either therapeutic hypothermia or standard care with normothermia, with target temperatures of 32°C to 34°C and 37°C, respectively. Hypothermia was maintained for 24 hours, and patients were then rewarmed passively over 8 hours. At 6 months, 55% percent of the hypothermia group had a favorable neurologic outcome, defined by cerebral performance category (CPC) score of 1 or 2 on a five-point scale, compared to 39% in the normothermia group. In addition, the 6-month mortality in the hypothermia group was 14% lower than it was in the normothermia group.

Despite a number of complications associated with hypothermia in the aforementioned trials, including coagulopathy, arrhythmia, hyperglycemia, and sepsis, the AHA guidelines adopted induced therapeutic hypothermia. The 2010 AHA guidelines recommended cooling of comatose adult patients with ROSC after out-of-hospital cardiac arrest due to pulseless ventricular tachycardia (VT)/VF to 32°C to 34°C for 12 to 24 hours (class 1, LOE B) and to consider hypothermia for most other comatose adult patients with ROSC after cardiac arrest regardless of initial rhythm (class IIb, LOE B).

In 2013, a large, well-performed trial was published that compared neurologic outcomes of patients after being randomized to two target temperatures. Nielsen et al. randomized 939 comatose adult patients with ROSC after out-of-hospital cardiac arrest from any rhythm to a target temperature of 33°C or 36°C. After 28 hours at target temperature, both groups were gradually rewarmed to 37°C, and between 36 and 72 hours, hyperthermia (>37.5°C) was aggressively avoided using fever control measures. At follow-up after 180 days, there were no significant differences between the two groups with respect to the composite outcome of death or poor neurologic function measured by the CPC or modified Rankin scale score.

From the results of the above-mentioned study, it seems that inducing hypothermia does not offer an advantage. However, preventing fever in these patients might pose a clinical benefit with regard to long-term neurologic outcome.

Up until recently, the AHA guidelines recommend cooling to 32°C to 34°C, with the support from the two landmark trials and several other studies that showed improved neurologic outcome for comatose survivors of VF cardiac arrest. In the 2015 updates, based on the most recent literature, the AHA strongly recommends that all comatose patients with ROSC should have temperatures maintained between 32°C and 36°C for at least 24 hours, after which fever should be actively prevented. The nomenclature of induced

hypothermia has been changed to “targeted temperature management” or TTM.

Targeted temperature management should be initiated as soon as possible in the emergency department in the appropriate post-ROSC patient. It should also be noted that prehospital initiation of hypothermia using cold intravenous fluids is no longer recommended in the most current guidelines. There is no optimal cooling method; however, current therapies include using cold saline, cooling blankets, and frequent application of ice packs. Available methods may be institution dependent. Core temperature should be continuously monitored using an esophageal thermometer, bladder catheter, or pulmonary arterial catheter if needed. After cooling (at which point the patient would typically be in the intensive care unit), fever should be avoided.

## KEY POINTS

- Two landmark trials in 2002 showed neurologic benefit from therapeutic hypothermia.
- The AHA adopted induced therapeutic hypothermia in 2005.
- A large trial in 2013 showed no difference in neurologic outcome between targeted temperatures of 33°C and 36°C.
- The most recent AHA updates recommend targeted temperature management between 32°C and 36°C for 24 hours post ROSC.
- Fever should be avoided as a part of targeted temperature management.
- Prehospital initiation of cold intravenous fluids is no longer advised.

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## **ACTIVATE THE CARDIAC CATH TEAM FOLLOWING SUDDEN CARDIAC ARREST—DON'T BE AFRAID TO CALL**

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**MATTHEW J. LEVY, DO, MSc, FACEP, FAEMS**

Emergency coronary artery angiography and percutaneous coronary intervention (PCI) is a mainstay of modern emergency medical care. It is well established that emergent PCI should be promptly performed in patients experiencing acute ST-segment elevation myocardial infarctions (STEMIs), provided it can be done so in a timely manner by a skilled and well-organized emergency cardiac team. The benefits of emergent PCI having been expanded to centers without cardiac surgery standby has had a profound effect in the form of decreased mortality and morbidity for those patients with an acute “culprit” obstructing coronary artery lesion and a very low complication rate.<sup>1</sup> There has also been a heightened interest in the benefit of emergent PCI in patients who have experienced sudden cardiac arrest (SCA), especially out-of-hospital cardiac arrest (OHCA) with return of spontaneous circulation (ROSC). A series of post-cardiac arrest patients with a suspected cardiovascular etiology noted that an emergently treatable coronary artery lesion was found in 96% of patients with ST elevation and in 58% of patients without STEMI elevation.<sup>2</sup>

The 2010 American Heart Association (AHA) Emergency Cardiac Care Guidelines recommend that postarrest patients with ROSC who possess electrocardiogram (ECG) evidence of STEMI receive emergent coronary angiography and prompt recannulation of any infarct-related artery.<sup>3,4</sup> The

2015 AHA guidelines reinforce the notion that acute coronary syndromes are a common etiology for SCA in adults without obvious extracardiac causes of arrest.<sup>5</sup> The 2015 AHA guidelines reference the 2015 ILCOR systematic review that examined immediate coronary angiography for patients after cardiac arrest. Two dozen observational studies reported either improved survival to hospital discharge or favorable neurologic outcome with emergency cardiac angiography in postarrest patient with STEMI. Interestingly, two observational studies reported improved survival to hospital discharge and improved neurologically favorable outcome associated with emergency coronary angiography in patients without ST elevation on initial ECG.

It has become more apparent that a subset of post–cardiac arrest patients with ROSC exists, who will not display STEMI on ECG but still have an acutely obstructing coronary artery lesion. The challenge is in the identification of which postarrest patient with ROSC without STEMI on their ECG will benefit from coronary angiography. It has been noted that SCA may not be accompanied by precedent symptoms or classic ECG manifestations of STEMI following ROSC, and further, many of the original studies examining emergent PCI in STEMI excluded cardiac arrest patients. The 2015 AHA ECC guidelines state that “*Emergency coronary angiography is reasonable for select (e.g., electrically or hemodynamically unstable) adult patients who are comatose after OHCA of suspected cardiac origin but without ST elevation on ECG (Class IIa, LOE B-NR).*” Many factors and considerations are suggested when considering which post–cardiac arrest patients with acute coronary syndromes without ST elevation should be considered for early coronary angiography, and include hemodynamic or electrical instability, comorbid conditions, presence of ongoing ischemia, and other patient characteristics.

Bottom line: It has become common practice for the post–cardiac arrest patient with ECG findings of STEMI to receive emergent coronary angiography. However, don’t assume that those ROSC patients with non-STEMI EKGs, and no apparent extracardiac cause of arrest, are not having an acute coronary artery obstruction. Don’t be afraid to make the call to the interventional cardiologist, discuss the case, and arrive at a plan that’s best for that patient.

## KEY POINTS

- Emergent PCI should be promptly performed in patients experiencing

acute STEMIs.

- A subset of post–cardiac arrest patients with ROSC exists, who will not display STEMI on ECG but will have an acutely obstructing coronary artery lesion, amenable to PCI.
- Factors to consider for PCI in ROSC patients who do not have an STEMI include hemodynamic or electrical instability, comorbid conditions, presence of ongoing ischemia, and other patient characteristics.
- When in doubt, discuss the case with the interventional cardiologist.

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## RUSH TO RESUSCITATION

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**DANIEL SHEETS, MD, MPH AND RANDALL T. RHYNE,  
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When a patient with undifferentiated shock presents to the emergency department, physicians are forced to make rapid management decisions based on limited information about the patient's history or recent course of illness. Ultrasound has emerged as an effective diagnostic tool that bedside physicians can use to get actionable information on the etiology of hypotension, as well as actively guide resuscitation and monitor potential improvement in the patient's clinical status. While many ultrasound protocols have been developed to aid in resuscitating critically ill patients, the Rapid Ultrasound in Shock or RUSH exam has risen in prominence due to its ability to rapidly and accurately diagnose the classification of shock. This exam takes a three-step bedside protocol to assess a critically ill patient's cardiovascular status ("the pump"), intravascular volume status ("the tank"), and vascular integrity ("the pipes"). This protocol integrates several familiar ultrasound applications that the emergency physician can perform in real time at the bedside to guide resuscitative efforts for patients in shock. The three steps of the RUSH exam are explained in further detail below.

### **“THE PUMP”—ASSESS CARDIOVASCULAR STATUS**

The first step in performing the RUSH exam on a patient in shock is a cardiac examination of “the pump.” The physician assesses the heart for pericardial effusion in the most dependent portion of the pericardium with a subxiphoid view. A large pericardial effusion may suggest cardiac tamponade, which in an unstable hypotensive patient indicates the need for

emergent pericardiocentesis to relieve obstructive shock. This investigation then proceeds to parasternal long and parasternal short axis views of the heart to assess cardiac contractility. A gross visual inspection of cardiac dynamics and wall excursion can be used to classify cardiac contractility as normal, reduced, hyperdynamic, or absent. A hyperdynamic heart with good contractility suggests hypovolemic or distributive shock that may benefit from a fluid bolus, while a hypodynamic heart with reduced contractility indicates cardiogenic shock that may benefit from vasopressors and inotropes. The final step in assessing “the pump” is to perform an apical four-chamber view, paying special attention to the size of the right ventricular. The right ventricle is normally smaller than the left ventricle. An acutely enlarged right ventricle should raise suspicion for obstructive shock caused by a massive pulmonary embolism.

## **“THE TANK”—ASSESS INTRAVASCULAR VOLUME STATUS**

The second step in performing the RUSH exam is to evaluate the patient’s intravascular volume status, that is, “the tank,” for fullness, leakiness, compromise, or overload, which is described below.

### ***“Tank Fullness”—Evaluation of the Inferior Vena Cava for Size and Collapsibility***

Using a phased-array or curvilinear probe, the fullness of the tank can be evaluated by measuring the inferior vena cava (IVC) width and assessing for collapsibility with inspiration. An IVC width  $< 2$  cm and collapsibility  $> 50\%$  of the vessel diameter during inspiration suggest decreased intravascular volume. In the setting of hypotension, these findings can indicate distributive or hypovolemic shock. After administering a fluid bolus, this exam may be repeated to determine any improvement in IVC diameter or collapsibility. Alternatively, an IVC width  $> 2$  cm in combination with decreased cardiac contractility may suggest cardiogenic shock. As a caveat, measuring IVC in an intubated patient becomes unreliable due to increased intrathoracic pressure.

### ***“Tank Leakiness”—Evaluation with Extended FAST Exam***

The next sequence is evaluating “the tank” for leakiness by performing an Extended Focused Assessment with Sonography in Trauma (eFAST) exam

to assess the abdomen for intraperitoneal free fluid and the bilateral lung bases for thoracic fluid, which could represent hemothorax in the appropriate clinical setting. Consideration should be given to conditions such as traumatic injuries, ruptured ectopic pregnancy, ruptured ovarian cyst, or ruptured abdominal aortic aneurysm that cause hemorrhagic shock.

### ***“Tank Compromise”—Evaluation for Pneumothorax***

After completing the abdominal portion of the eFAST exam, evaluation of the “the tank” progresses to an assessment of the lungs with a linear probe. The presence of bilateral lung sliding and comet tail artifact rules out hypotension due to tension pneumothorax causing obstructive shock.

### ***“Tank Overload”—Evaluation for Pulmonary Edema***

The phased-array or curvilinear probe may then be used to assess the thorax for B-lines, which if present bilaterally are suggestive of pulmonary edema due to cardiogenic shock.

## **“THE PIPES”—ASSESS VASCULAR INTEGRITY**

The final step of the RUSH exam on a hypotensive patient is to evaluate the vascular system or “the pipes.” Using a low-frequency probe, the proximal, middle, and distal abdominal aorta should be examined for a diameter >3 cm, which is indicative of an aneurysm. The presence of an aortic aneurysm may suggest a ruptured aortic aneurysm as the etiology of hypotension. The lower extremity veins can also be assessed using a linear probe for the presence of deep vein thrombosis. This study should investigate the common femoral vein down through its bifurcation and the popliteal vein in the popliteal fossa for the presence of a thrombus. Deep vein thrombosis in the lower extremities may represent massive pulmonary embolism and obstructive shock as a possible etiology of hypotension. The diagnosis may be supported by the presence of an enlarged right ventricular and right heart strain visualized on the ultrasound.

The undifferentiated hypotensive patient can present a challenge for the emergency physician, requiring decisive action with limited information during resuscitation. A RUSH exam can provide a quick and noninvasive evaluation of “the pump,” “the tank,” and “the pipes” to help determine the etiology of hypotension. Be inspired to perform bedside ultrasound for your next hypotensive patient to rapidly and easily gain valuable clinical information and help guide management of the resuscitation.

## KEY POINTS

- RUSH to resuscitation in the undifferentiated hypotensive patient.
- Evaluate “the pump” for pericardial effusion, contractility, and signs of right ventricular strain.
- Evaluate “the tank” for IVC size and collapsibility, intraperitoneal free fluid, pleural fluid, pneumothorax, and pulmonary edema.
- Evaluate “the pipes” for aortic aneurysm and deep vein thrombosis.
- Reevaluate after interventions to assess changes in volume status and cardiac contractility.

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# DO NOT DELAY THE ADMINISTRATION OF EPINEPHRINE FOR PATIENTS WITH ANAPHYLAXIS

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NOUR AL JALBOUT, MD

Anaphylaxis is a serious systemic IgE-dependent immunologic hypersensitivity reaction, potentially fatal without early detection and management. Anaphylaxis tends to be underrecognized and undertreated with a lifetime prevalence based on international studies estimated to be at 0.05% to 2%. Based on the World Allergy Organization guidelines published in 2011, anaphylaxis is diagnosed if *any* of the following three criteria are met:

- Onset of illness involving skin or mucosal tissue with respiratory compromise or reduced blood pressure
- An exposure to a likely allergen with the development of two of the following: skin or mucosal tissue involvement, reduced blood pressure, respiratory compromise, or persistent gastrointestinal (GI) symptoms
- Reduced blood pressure after exposure to a known allergen

Main allergy triggers differ by age group and geographical region. Food is the most common precipitant of anaphylaxis in pediatric and young adult populations, while insect stings and medications are more common precipitants in middle-aged and elderly adults. Be sure to ask about all exposures and events in the hours preceding the onset of symptoms. Remember that different patient factors contribute to the severity of anaphylaxis, namely, age, comorbidities, and concurrent medications.

As with any patient, the approach to treatment must be systematic. Start

by removing any exposure to the trigger if it is still present, while rapidly addressing the patient's airway, breathing, and circulation. If anaphylaxis is suspected, immediate treatment with intramuscular (IM) epinephrine is indicated. In the setting of anaphylaxis, the pediatric dose of epinephrine is 0.01 mg/kg of a 1:1,000 (1 mg/mL) solution. The adult dose is generally 0.3 to 0.5 mg of 1:1,000 epinephrine solution (0.5 mg is the maximum dose). Note that 0.3 mg is the dose contained in a standard adult epinephrine autoinjector. Depending on the severity of symptoms and the patient's response to the initial dose, epinephrine can be repeated every 5 to 15 minutes, as needed. In patients who do not respond to IM epinephrine, assess the intravascular fluid status carefully before progressing to intravenous epinephrine. However, if circulatory shock has developed and cardiac arrest is imminent, administer epinephrine intravenously or intraosseous by slow infusion (1 to 10 µg/min for adults, and 0.1 µg/kg/min for children), titrating it according to hemodynamic parameters. Complications from IM epinephrine are rare; patients who are elderly or have known cardiovascular disease may also benefit from receiving epinephrine. The benefits from reversing cardiovascular collapse outweigh the risks of hypertension and increased cardiac demand.

Evidence favors prompt epinephrine injection over the use of antihistamines and glucocorticoids for the treatment of anaphylaxis. Why does epinephrine work? The therapeutic effect of epinephrine stems from its mechanism of action, as it directly reverses the pathophysiology behind anaphylaxis. Namely, epinephrine has alpha-1 adrenergic agonist effects, increasing peripheral vascular resistance while decreasing mucosal edema. These critical two effects address potentially lethal hypotension and airway compromise. In addition, epinephrine has beta-1 adrenergic agonist effects increasing heart rate and cardiac contractility and beta-2 adrenergic agonist effects leading to bronchodilation and decreased release of inflammatory mediators from mast cells and basophils.

Failure to inject epinephrine promptly is associated with severe cardiopulmonary compromise and biphasic anaphylaxis with recurrence of symptoms in up to 72 hours. Yet, it is crucial to watch for critical side effects of epinephrine. Rarely, epinephrine may lead to ventricular arrhythmias, acute coronary syndrome, pulmonary edema, and intracranial hemorrhage. Bear in mind that serious adverse effects occur most commonly after rapid intravenous bolus injections or incorrect dosing.

Are there any second-line treatments available? Evidence for the administration of second-line medications such as antihistamines, beta-2 adrenergic agonists, and glucocorticoids is extrapolated mainly from their use in treating urticaria (treated by antihistamines) and acute asthma (treated

by beta-2 adrenergic agonists and glucocorticoids). Do not focus on those medications if they will delay the prompt administration of epinephrine. Antihistamines (H<sub>1</sub> and H<sub>2</sub> blockers) are potentially beneficial to relieve the patient's itching, flushing, and urticaria. The concerns with using H<sub>1</sub> blockers include their potential to cause somnolence and their slow onset of action. Little evidence exists for the concurrent use of H<sub>2</sub> blockers. Unlike epinephrine, beta-2 agonists help to relieve wheezing, coughing, and shortness of breathe by relaxing smooth muscle. However, beta-2 agonists have no effect on the upper airway edema since no smooth muscles are present there. Systemic glucocorticoids take several hours to work. Their role is to potentially relieve protracted anaphylaxis symptoms and prevent biphasic anaphylaxis, although these effects have not been proven.

Deciding on the disposition of the patient poses another challenge for clinicians. This decision should be based on the severity of the patients' symptoms and their prompt response to initial interventions. Patients with moderate respiratory or cardiovascular compromise should be monitored for at least 4 to 6 hours, whereas patients with severe or protracted anaphylaxis may require prolonged monitoring and inpatient admission, ranging from acute short-term to intensive care. Discharged patients must be informed and educated about recurring symptom(s). Patients should be advised to follow up with their primary care physician and an allergist to confirm the trigger since the key to preventing symptoms is to avoid the allergen. Explanation should include a description of epinephrine as well as instructions for when and how to self-administer this medication, emphasizing the importance of staying equipped with an epinephrine autoinjector. Discharge instructions should include a prescription for an epinephrine autoinjector if appropriate and also consider a refill for this potentially lifesaving medication. Beware that this device may be prohibitively expensive for the patient and may require social work involvement.

Due to its potential for serious morbidity and mortality, it is essential for emergency medicine clinicians to rapidly recognize and treat anaphylaxis. Furthermore, physicians have a duty to educate patients and their families about how to prevent anaphylaxis and to treat symptomatic patients with lifesaving epinephrine when necessary.

## KEY POINTS

- Anaphylaxis in patients is common; recognize its effect on potential organs including skin, mucosa, heart, lungs, and the GI tract and treat

promptly.

- Immediately address the airway, breathing, and circulation issues, and keep in mind that patients must be monitored for at least 4 to 6 hours.
- The recommended dose of epinephrine is 0.01 mg/kg of 1:1,000 solution intramuscular (IM). An adult dose generally ranges between 0.3 and 0.5 mg IM.
- Do not delay administering epinephrine when also giving antihistamines or glucocorticoids.
- Educate patients about avoiding allergy triggers and how to use epinephrine autoinjectors.

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## PUTTING ON THE SQUEEZE... VASOPRESSORS

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In critically ill patients, utilization of vasoactive medications can potentially be a lifesaving intervention. It is important for the emergency clinician to have an understanding of the pathophysiologic principles of the various types of shock as well as the pharmacologic mechanism of action of the drugs used to treat shock. The understanding of these physiologic principles will aid the emergency clinician in the selection, initiation, and titration of vasopressors while avoiding common pitfalls associated with their use. Shock is a state of hypoperfusion due to inadequate oxygen delivery at the tissue level and increased oxygen consumption, manifested by circulatory failure. The four types of shock include distributive, hypovolemic, cardiogenic, and obstructive. Distributive shock is characterized by profound systemic vasodilation seen in several forms including septic, anaphylactic, neurogenic, and acute adrenal insufficiency. Hypovolemic shock occurs when intravascular volume is depletion through hemorrhagic losses or nonhemorrhagic losses (GI, renal, skin, or third spacing). In cardiogenic shock, forward flow is inadequate due to intracardiac pump failure. This can be a result of significant myocardial ischemia, atrial/ventricular arrhythmia, or mechanical failure (i.e., valvular insufficiency or defects). Obstructive shock occurs when extracardiac causes of cardiac pump failure result in poor right ventricular output. In conditions such as massive pulmonary embolism and pulmonary hypertension, right ventricular failure occurs as a result of inability of the right ventricle to generate pressures high enough to overcome the pulmonary vascular resistance. In cardiac tamponade and tension pneumothorax, impaired venous return (i.e., preload) is the primary mechanism. The four categories of shock provide a framework for

understanding the etiology of the various types of shock; however, it is important to understand that they are not exclusive. Patients may present with a form of combined or undifferentiated shock, and the emergency clinician must provide prompt vasoactive support to prevent irreversible organ dysfunction and death.

Vasopressors are a class of drugs that elevate mean arterial pressure (MAP) by inducing systemic vasoconstriction. These include norepinephrine, vasopressin, epinephrine, phenylephrine, and dopamine (dose dependent). It is important to differentiate vasopressors from inotropes, which increase cardiac contractility. However, some drugs have both vasopressor and inotropic effects. Understanding the location of the receptor on which the vasopressor acts will help further classify each drug and aid in selection of vasopressors.

Selection of vasopressors should incorporate current medical literature and knowledge of the physiologic principles of the types of shock. The most recent Surviving Sepsis guidelines have recommended norepinephrine as the agent of choice with epinephrine or vasopressin as second line. In patients with cardiogenic shock, norepinephrine followed by epinephrine, and consideration of dopamine at lower doses may be used. In neurogenic shock, norepinephrine should be regarded as first-line agent because of its ability to provide both vasoconstrictive and chronotropic effects. Epinephrine is still the preferred drug in the treatment of anaphylactic shock because of its vasoconstrictive, inotropic, chronotropic, and bronchodilator properties. Obstructive shock due to massive pulmonary embolus should be treated with norepinephrine or epinephrine due to their ability to increase preload by vasoconstriction, improve inotropy, and increase MAP. In hemorrhagic shock, vasopressors are generally not indicated. Rather the focus should be on resuscitation with blood products.

Vascular access should be considered when choosing to start vasoactive drugs. While practice patterns and hospital policies may vary, in the critically ill patient, initiation of vasopressors should not be delayed if the patient has adequate peripheral access (in AC and 20 g or greater). Once initiated in a peripheral IV, vigilant extremity checks should be started. If the patient remains dependent on vasopressors for hemodynamic stability, a central line should be placed. If unable to place central access or obtain a reliable peripheral IV, consider an interosseous line.

Deciding when to initiate vasopressors depends on the clinical situation that you are facing and the etiology of the shock affecting your patient. In septic and nonhemorrhagic hypovolemic shock, if an IV fluid challenge (30 mL/kg) has failed to restore normotension, vasoactive therapy should be

initiated. This differs from obstructive shock due to pulmonary embolus, in which only small volume of IV fluid (500 to 1,000 cc) should be given prior to starting vasopressors. Too much volume resuscitation in massive pulmonary embolus overdistends the right ventricle, causing diastolic compression of the left ventricle, therefore reducing cardiac output. Each individual case will need to be accessed when deciding to initiate vasopressor therapy.

Titration of vasoactive agents should be clear, objective, and evidence based. Orders should be written with a starting dose (weight based), titration dose with a time interval, a goal measure, and maximum dose. For example, for norepinephrine, start infusion at 0.02 mcg/kg/min; titrate q5min by 0.02 mcg/kg/min, for goal MAP > 65, with the maximum dose at 3 mcg/kg/min. In order to determine the need for titration of our vasopressor, we must determine its effectiveness through objective measures of end organ perfusion. Literature has proven that a MAP > 65 preserves tissue perfusion and a urine output of 0.5 cc/kg/hr is indicative of adequate renal perfusion.

Circulatory shock is associated with increased morbidity and mortality. Prompt recognition and vasopressor therapy should be initiated in refractory hypotension despite adequate resuscitation. Selection of therapy should be based on the suspected underlying etiology of shock. Titration of the agent should target objective indices of perfusion (i.e., MAP > 65, CPP > 80, etc.). By providing prompt, evidence-based vasoactive therapy, the emergency clinician can maintain end organ perfusion and prevent irreversible organ failure, while simultaneously aiming to identify and treat the underlying cause.

## KEY POINTS

- The patient should be adequately volume resuscitated prior to initiating vasopressors.
- Lack of central venous access should not delay initiation of vasopressors if there is adequate peripheral IV access.
- Orders should be written with a starting dose (weight based), titration dose with a time interval, a goal measure, and maximum dose.
- Adrenal crisis should be considered in patients on high-dose or multiple vasopressors.
- Vasopressor selection should incorporate current medical literature and knowledge of the physiologic principles of the types of shock.

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# **HOW MUCH IS ENOUGH? TRANSFUSIONS IN THE BLEEDING PATIENT: DON'T FORGET THE REST OF THE BLOOD**

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To stabilize the exsanguinating patient is one of the classic missions in emergency medicine. Recent study has provided evidence-based guidance of massive transfusion (MT), establishing that early balanced resuscitation improves both patient outcomes and stewardship of our blood banks, leading to the widespread use of massive transfusion protocols (MTPs). MT has been studied most extensively in the setting of trauma, but its use is supported in other hemorrhagic conditions including vascular and obstetric catastrophe. The management of MT, therefore, is no longer the concern of only the trauma surgeon and anesthesiologist; knowledge of MTP is essential to emergency practice in all types of facilities, from the tertiary center with high volumes of trauma to the rural hospital receiving the peri-arrest patient who requires stabilization for transfer.

Patients in hemorrhagic shock suffer from a variety of physiologic complications. Even before entering the critical care bay, they often are coagulopathic (as a result of either bleeding or the injury that led to the bleeding), hypothermic, and acidotic: the “lethal triad.” This vicious cycle can continue as the remaining clotting factors become ever more dysfunctional at suboptimal temperature and pH, leading to worsening coagulopathy, hypothermia, and acidosis. Bleeding patients often require transfusion, not merely volume, to survive long enough for definitive

intervention.

Massive transfusion historically is defined as >10 units of packed red blood cells (PRBC) in 24 hours. MT is fraught with complications beyond the original insult. Dilution by PRBC of the remaining coagulation factors and platelets can lead to continued bleeding and need for more PRBC. Metabolic derangements can occur and often includes hypocalcemia, hyperkalemia, and alkalosis from metabolism of citrate (added to anticoagulate stored blood). Other postoperative complications can occur such as prolonged ICU stay, infection, and organ failure.

Balanced resuscitation, also known as “damage control,” mitigates many problems associated with traditional MT and comprises balanced ratios of blood products, permissive hypotension, and the minimization of crystalloid. “Survivor bias” (patients who did not survive long enough to receive ten units of PRBC were not counted as recipients of MT) undermined earlier studies, but later prospective trials correct for this and demonstrate that patients who go on to receive MT have better outcomes when balanced resuscitation is implemented in the first 6 hours of care, including decreased mortality at 24 hours and 30 days, fewer total blood products used, more ventilator-free days, and fewer complications such as multisystem organ dysfunction and infection. Many of these benefits are lost if the damage-control approach is implemented after the first 6 to 24 hours of care.

The best approach to balanced resuscitation is debated, with some authors advocating goal-directed replacement of coagulants, that is guided by prothrombin time or thromboelastography. While arguably more precise, this approach may require resources not universally available. The alternative is to address coagulopathy proactively, with the early units of PRBC. Upon activation of the MTP, a facility’s blood bank delivers a bundle of plasma, platelets, and PRBC for transfusion in a specific ratio and order; the teams maintain contact regarding anticipated needs. The particular ratio varies among institutions; 1:1:1 has support in the literature and is closest to whole blood. Whole blood, ideal in many regards, has limited availability in most civilian areas, where blood storage and distribution is facilitated by separation into components.

Researchers have attempted to better predict who will need MT to enable timelier implementation of balanced resuscitation. The Trauma-Associated Severe Hemorrhage (TASH) score requires laboratory data and a weighted, logarithmic calculation, making its use in the critical care bay cumbersome. The Assessment of Blood Consumption (ABC) score is simplified and limited to data immediately available on initial evaluation: systolic blood pressure (SBP) < 90, pulse > 120, positive bedside sonography, and

penetrating mechanism. One study found it equivalent to physician gestalt, which is unsurprising given its origin (physicians known to be early activators of MTP were asked why, and their answers informed the ABC tool). The revised Massive Transfusion Score (rMTS) combines portions of ABC and TASH: SBP < 90, base deficit  $\geq$  6, international normalized ratio (INR) > 1.5, hemoglobin < 11, and temperature < 35.5; its validation study showed noninferior sensitivity and improved specificity versus the previous tools, but overall accuracy remained moderate, ranging from 55% to 70% depending on the cutoff. Its creators note that pelvic trauma is a scenario vulnerable to false-negative screening with both rMTS and physician gestalt. In the absence of a widely accepted objective trigger for MTP, many trauma centers leave activation to attending discretion.

The last decade of research has underscored that exsanguinating patients do poorly when overresuscitated with PRBC or crystalloid. If a patient needs more than two units of red blood cells, the other components will be needed shortly—don't forget the rest of the blood!

## KEY POINTS

- Provide balanced resuscitation.
- Think about MTPs early. The decision not to activate should be based on clinical assessment and not the failure to consider it.
- If your institution lacks an MTP, consider a 1:1:1 ratio of platelets, plasma, and red cell units as a guide.
- Consider MTP in nontrauma settings.

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# **FLUID THERAPY: BEWARE OF (AB)NORMAL SALINE—CHOOSE YOUR RESUSCITATION FLUIDS CAREFULLY**

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For an average adult, water accounts for 60% of total body mass. However, this percentage decreases as the percentage of fat increases since fat has a lower water content than lean tissue. For a normal, healthy 70-kg adult, daily water intake should be between 2,000 and 3,000 mL to make up for urinary and insensible losses. Total body water is partitioned between intracellular and extracellular fluid compartments. Water moves from the compartment of low osmolality to that of higher osmolality until the fluids on either side of the barrier share the same osmolality. If this redistribution of water is too excessive or rapid, then the corresponding changes in cell volume can lead to cellular dysfunction or injury. This disruption in fluid balance can be from hemorrhagic or nonhemorrhagic volume losses.

Some of the most commonly treated conditions in the emergency department (ED) requiring intravenous (IV) fluid resuscitation include trauma, gastrointestinal (GI) losses, sepsis, diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS), hyponatremia, rhabdomyolysis, and burns. Resuscitation fluids should be chosen so as to prevent further cellular injury while restoring homeostasis.

Fluids are classified according to molecular weight and oncotic pressure. Crystalloids have a lower molecular weight and lower oncotic pressure compared to colloids. Colloids preserve oncotic pressure and their vascular

retention makes them more efficient volume expanders with a longer duration of action than crystalloids. However, when dosed appropriately, isotonic crystalloid solutions like lactated Ringer's (LR) or normal saline (NS) are equally effective volume expanders. Therefore, with few exceptions, there appears to be no proven clinical superiority to using colloids over crystalloids. Considering the significant cost advantage and availability, crystalloid solutions are the most commonly used resuscitation fluid in the ED. A table listing the different types of each fluid and their contents can be found in [Table 21.1](#).

| <b>TABLE 21.1 TYPES OF FLUID</b> |                       |                      |                       |                       |                |               |           |
|----------------------------------|-----------------------|----------------------|-----------------------|-----------------------|----------------|---------------|-----------|
| <b>FLUID</b>                     | <b>Na<sup>+</sup></b> | <b>K<sup>+</sup></b> | <b>Ca<sup>+</sup></b> | <b>Cl<sup>-</sup></b> | <b>LACTATE</b> | <b>MOSM/L</b> | <b>PH</b> |
| NS                               | 154                   |                      |                       | 154                   |                | 308           | 5         |
| LR                               | 130                   | 4                    | 2.7                   | 109                   | 28             | 273           | 6.4       |
| 3% NS                            | 513                   |                      |                       | 513                   |                | 1027          | 5         |
| 0.45% NS                         | 77                    |                      |                       | 77                    |                | 154           | 5         |
| Albumin                          | 145                   |                      |                       | 95                    |                | 300           |           |

Total body fluid loss due to hypovolemia can be from trauma, hemorrhage, or GI losses. Patients experiencing uncontrolled hemorrhagic shock should be treated with deliberate hypotensive fluid resuscitation, as overaggressive resuscitation can exacerbate their ongoing bleeding by interrupting formed thrombus. Efforts should be focused on getting the patient to surgery to identify and stop the bleed. For patients in controlled hemorrhagic shock where the bleeding site has been identified and stopped, fluid resuscitation is aimed at normalizing their hemodynamics. In both cases, the choice of fluids is pRBCs, FFP, and platelets given at a ratio of 1:1:1.

Patients with GI losses from profuse vomiting, gastric outlet obstruction, and nasogastric suctioning develop a hypochloremic metabolic alkalosis. In these patients, the best choice is NS as it contains a higher concentration of chloride. Patients with diarrhea requiring IV fluid resuscitation should receive LR as it better corrects the hyperchloremic metabolic acidosis they can develop.

Patients in septic shock require aggressive IV fluid resuscitation. The preferred fluid is crystalloid as randomized trials and meta-analyses have found increased cost with no clear benefit with the use of colloids.

Patients in DKA or HHS require careful electrolyte and fluid

replacement and the choice of resuscitation fluid depends upon the patient's initial state of hydration, blood pressure, and sodium, potassium, and glucose concentrations. In general, the recommended initial resuscitation fluid is NS. The suggested amount is 1 to 1.5 L given during the first hour, adjusted accordingly depending upon the patient's clinical state. Subsequent fluids should be either 0.45% NS or LR to prevent hyperchloremic metabolic acidosis. Dextrose should be added when the serum glucose level reaches 200 mg/dL in DKA or 250 to 300 mg/dL in HHS. Adequate fluid resuscitation will result in an increased response to insulin. Cerebral edema may develop if the osmolality is reduced too rapidly; however, this is more of a concern in the pediatric patient.

Treatment of hyponatremia varies based on duration, sodium level, volume status, and symptoms. The overall goal is to increase serum sodium by 4 to 6 mEq/L over the first several hours, but not to exceed 8 mEq/L in any 24-hour period to avoid the risk of central pontine myelinolysis. Acute, severe hyponatremia (sodium < 120 mEq/L) presenting with altered mental status, seizures, or coma should be treated emergently with 3% hypertonic saline with initial 100-mL bolus given over 10 minutes followed by another 100 mL over the next 50 minutes; then reassess. Each 100-mL bolus will raise sodium by 2 to 3 mEq/L. Asymptomatic patients with acute or hyperacute severe hyponatremia should receive 3% hypertonic saline at a rate of 0.5 to 2 mL/kg/h. For patients with moderate to severe hyponatremia from SIADH or in hypervolemic state, the initial first-line treatment is fluid restriction with a goal of 500 mL/d below the 24-hour urine volume. NS is not the preferred emergent choice as it will raise the serum sodium slower than hypertonic saline and patients with SIADH do not respond well to it.

Rhabdomyolysis requires early and aggressive fluid resuscitation to prevent acute kidney injury (AKI) and increase urinary potassium excretion. The initial treatment is NS at a rate of 1 to 2 L/h. Patients who have a stable plasma CK (creatinine kinase) < 5,000 units/L do not require IV fluid, as studies have shown the risk of AKI is low. The use of chloride-restrictive fluid (LR) instead of NS has been reported to decrease the incidence of AKI; however, there are no studies that directly compare the efficacy and safety of different types and rates of fluid administration in this setting.

Patients with superficial deep and full-thickness burns >30% of total body surface area should receive fluid resuscitation with LR. The Parkland formula is used to guide acute resuscitation, with continued reassessments using urine output and hemodynamics to make any alterations as needed. After the first 8 hours, if the patient is continuing to require large volumes of fluid, albumin can be substituted for 1/3 of the crystalloid, remembering to adjust the dose of albumin where 1 cc of albumin is equivalent to 3 cc of

crystalloid.

The major complications from improper management of IV fluid resuscitation are inadequate or excessive volume replacement, electrolyte abnormalities, and hematologic complications.

## KEY POINTS

- With few exceptions, there appears to be no proven clinical superiority to using colloids over crystalloids.
- Patients with GI losses from profuse vomiting, gastric outlet obstruction, and nasogastric suctioning develop a hypochloremic metabolic alkalosis. In these patients, the best choice is normal saline.
- Patients with diarrhea requiring IV fluid resuscitation should receive LR as it better corrects the hyperchloremic metabolic acidosis they can develop.
- Rhabdomyolysis requires early and aggressive fluid resuscitation to prevent AKI and increase urinary potassium excretion. The initial treatment is NS at a rate of 1 to 2 L/h.
- DKA or HHS requires careful electrolyte and fluid replacement; the recommended initial resuscitation fluid is NS at 1 to 1.5 L given during the first hour, adjusted accordingly depending upon the patient's clinical state.

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# ECMO

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Extracorporeal membrane oxygenation (ECMO) is an increasingly popular technology that is used as a modified cardiopulmonary bypass in order to oxygenate blood externally. First demonstrated in the 1970s, it has recently gained popularity as a rescue therapy for patients in severe respiratory failure, circulatory failure, and prolonged cardiac arrest. While multiple studies have established ECMO as an effective rescue intervention in pediatric populations with cardiopulmonary failure, large RCTs have yet to establish the same utility in adults, and its clinical effectiveness is still uncertain. Advancements in ECMO technology and widespread use during the 2009 to 2010 H1N1 outbreak have led to a rise in use.

There are two basic configurations for ECMO: venovenous (VV) and venoarterial (VA). VV ECMO accesses and returns blood from the venous system and provides nonpulmonary gas exchange, and thus is most commonly used for gas exchange and respiratory support. VA ECMO accesses the venous system and returns to the arterial system and is used for circulatory support in prolonged cardiac arrest and cardiac failure. Extracorporeal cardiopulmonary resuscitation (ECPR) utilizes VA ECMO as a method to provide gas exchange and circulatory support. When conventional algorithms fail, ECMO may represent a rescue therapy for patients in extremis.

## PROPOSED PHYSIOLOGY

ECMO offers the possibility of supporting patients with nonsurvivable heart and lung pathology when conventional maneuvers are failing and allowing increased time to treat underlying illness. Utilizing ECMO allows gas exchange despite lack of native lung function, potentially allowing further

lung-protective ventilation strategies to be used. In patients with refractory cardiac failure, VA ECMO provides oxygenated blood to the arterial system in a nonpulsatile fashion, supplementing cardiac output.

## BASIC CONFIGURATION

The configuration of ECMO is highly variable, and several pump-drive and pumpless systems exist. Essentially, the basic configuration consists of specialized cannula, circuit tubing, a centrifugal pump, heat exchanger, bladder reservoir, and a membrane that oxygenates blood and removes carbon dioxide. The tubing runs to and from the patient, and blood is driven through the oxygenation membrane by the centrifugal pump head, warmed to an appropriate degree, and returned to the patient. This is done under systemic anticoagulation, in which unfractionated heparin is most commonly used.

## BASIC TECHNIQUE

VV ECMO may be performed in a variety of ways. Typically, large cannulas are used and inserted via the Seldinger technique; deoxygenated blood is removed by accessing the femoral vein and advancing the cannula to the inferior vena cava. Return access is gained through the right internal jugular, thus returning oxygenated blood to the right heart. VA ECMO is typically performed at the femoral sites bilaterally. A venous catheter is inserted into the femoral vein, and an arterial catheter is inserted into the contralateral femoral artery. Care must be taken to avoid limb ischemia, as the arterial access site can potentially occlude the entire femoral artery. Initial cardiac output targets of 1.5 to 2.0 L/min are appropriate with titration to 3.0 to 6.0 L/min. Initiation of ECMO by ED physicians with meaningful long-term patient survival has not been demonstrated, though there have been case series demonstrating the feasibility of emergency physicians starting ED ECPR with promising results.

## INDICATIONS

- *Toxic overdose*—While the literature in treating overdoses with VA ECMO is still growing, there are case series demonstrating its usefulness in mostly cardiovascular medications—most specifically calcium and beta-blockers. In these cases, VA ECMO began early (<1 hour), and typical duration of therapy was <1 week.
- *Hypercarbic respiratory failure*—VV ECMO in this setting allows the

removal of carbon dioxide while also allowing lung-protective ventilation strategies. Patients who stand to gain the most from this are those with acute respiratory distress syndrome (ARDS) by resolving hypercarbia, decreasing the acidosis, and reducing the complication of barotrauma.

- *Cardiogenic shock*—Loss of mechanical pump function will inevitably cause multiorgan dysfunction, and in situations where vasopressor and inotropic support are inadequate, VA ECMO remains a viable rescue therapy. The goal of VA ECMO in this case is to augment cardiac output and reduce the dependence of vasopressive medications.
- *Pulmonary embolism*—VV ECMO allows for patients with pulmonary embolism (PE) with hemodynamic compromise a bridge to thrombolysis and catheter-based interventions.
- *Prolonged cardiac arrest*—The goal of using VA ECMO in a patient with prolonged cardiac arrest is to restore circulation. Like most ECMO topics, evidence for the utility of ECMO in cardiac arrest is still growing; however, early observation trials are promising. Most notably, ECMO has the most effectiveness when performed in patients with an etiology that is likely reversible. Keep in mind however that AHA Guidelines for CPR indicate that there is inadequate support to recommend the routine use of ECMO.

## RISKS

There are a fair amount of risks that are inherent to ECMO. Risks include artery occlusion leading to limb ischemia, hemorrhage secondary to anticoagulation, thrombosis and embolization from the centrifugal pump, and air embolism if the tubing is not primed appropriately. Another caveat is that ECMO should not be expected to continue indefinitely, and with that in mind, candidates for ECMO should be selected very carefully. Suggested exclusion criteria for initiation of ECMO include history of severe neurologic dysfunction, intracranial hemorrhage, terminal malignancy, uncontrolled bleeding, and severe peripheral vascular disease.

## KEY POINTS

- ECMO can be used as rescue therapy for cardiopulmonary support.
- At this time, there have been very few studies proving the efficacy of ECMO, especially in the setting of prolonged, out of hospital arrest.
- The basic types of ECMO are venovenous (VV) and venoarterial

(VA). Both have different benefits and indications, but are similar in setup: cannula for vascular access, and then tubing, pump, membrane oxygenator, and warmer.

- Potential considerations for ECMO: hypercarbic respiratory failure, cardiac failure, refractory reversible cardiac arrest, and PE with hemodynamic compromise.
- Risks of ECMO are varied and include air embolism, limb ischemia, thrombosis, and hemorrhage after anticoagulation.

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# **NEEDLE THIS: DO NOT ASSUME THAT NEEDLE DECOMPRESSION OF A TENSION PNEUMOTHORAX IS RELIABLE AND EFFECTIVE**

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**BAHRENEGASH GETACHEW, MD**

The knee-jerk reaction to signs and symptoms of tension pneumothorax is to obtain a large-bore needle for an immediate decompression at the 2nd intercostal space in the midclavicular line or at the 4th to 5th intercostal space in the midaxillary line. Regardless of the approach, needle thoracostomy for tension pneumothorax is a technique that every emergency medicine physician must master and be ready to employ. However, is this the most effective and reliable intervention?

The old mantra that the diagnosis of tension pneumothorax had to be established prior to a chest x-ray is becoming obsolete. The initial recommendation was based on logistics of the procedure and anatomy access and was probably not evidence based. Recently, the trauma literature has identified several pitfalls in utilizing needle decompression. Stevens et al. (2009) discovered a 50% success rate of this procedure, particularly in the prehospital settings.

A notable drawback of relying on needle thoracostomy is the variability of a patient's body habitus, which can make it difficult to access the pleural space effectively. Obesity rates have certainly added to the complexity; however, even a muscular individual with hypertrophied pectoral muscles could make the procedure more challenging. A study in the trauma literature by Powers et al. (2014) demonstrated a direct relationship between BMI and

chest wall thickness. For example, an average BMI of 29 corresponded with a 6.2-cm thickness at the 2nd intercostal space. Considering a 16 to 18g angiocatheter measures at 4.77 cm, the risk of failure is significant.

We often think of needle decompressions for tension pneumothorax as being both diagnostic and therapeutic. The sound generated from the rapid release of trapped air is generally the diagnostic tool, which can be a deceiving notion. There have been case reports describing a “hissing” sound from bullae in a patient with severe COPD who presented with acute respiratory distress. On the other end of the spectrum, if the needle is improperly implanted in subcutaneous tissue, sound may not be produced, thus falsely reassuring the unsuspecting physician.

The clinical situation of a tension pneumothorax is also vital to appraise when approaching the treatment for this entity. The distinction of a ventilated patient versus one who is not intubated is critical. The pathophysiology and compensatory mechanisms of tension pneumothorax in these patient populations differ greatly. A ventilated patient who develops pneumothorax will drastically and dramatically decompensate due to supraphysiologic pressures being provided artificially. A small pneumothorax can quickly transform into tension physiology, necessitating needle thoracostomy as the immediate first step. However, there is a growing body of evidence that the treatment of a nonventilated patient with tension pneumothorax without immediate hemodynamic instability may fall in a wide clinical spectrum with a more robust compensatory capability. These patients may benefit from diagnosis verification and a more definitive initial treatment intervention with tube thoracostomy.

Blind needle decompression is not without risks, no matter how well intended or anatomically accurate the physician is. Significant vascular, cardiac, and lung parenchyma injuries have been reported. Other common complications include iatrogenic hemothorax and pneumothorax. Once the needle is inserted, the physician has committed himself to tube thoracostomy, regardless of the final diagnosis.

The bottom line is that tension pneumothorax is a truly life-threatening event. When suspected, or the allusive diagnosis is actually obtained, immediate intervention is necessary. Needle decompression has a pivotal role, especially when hemodynamic instability accompanies this diagnosis. However, it's important to keep in mind that the definitive treatment of a tension pneumothorax is a chest tube.

## KEY POINTS

- Ensure the length of the needle is appropriate for the patient's body habitus.
- Ventilated patients decompensate faster when tension physiology is present.
- The presence or absence of the “hissing” sound can be deceiving.
- Tube thoracostomy is the definitive treatment.
- Needle decompression must *always* be followed by a chest tube.

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## RESUSCITATIVE THORACOTOMY

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Resuscitative thoracotomy is perhaps the most dramatic—and certainly the most invasive—procedure in the emergency physician's armamentarium. When performed on the appropriate patient for the appropriate indications, this procedure has the potential to produce striking results.

According to the American College of Surgeons Committee on Trauma, a resuscitative thoracotomy is indicated for patients who have suffered a penetrating thoracic injury and are observed by health care personnel (either prehospital or emergency department) to have objective signs of life prior to cardiopulmonary arrest and loss of vital signs. Objective signs of life include pupillary response, spontaneous ventilation, carotid pulse, measurable or palpable blood pressure, extremity movement, cardiac motion on ultrasound, and/or cardiac electrical activity. The procedure may be considered in patients sustaining penetrating nonthoracic injury, though the likelihood of survival in this instance is low. Due to its very low rate of survival in patients who have suffered blunt traumatic injury, resuscitative thoracotomy should be considered only rarely for victims of blunt trauma with vital signs in the emergency department who subsequently suffer cardiopulmonary arrest witnessed by emergency department personnel.

Outcomes following resuscitative thoracotomy are dependent upon multiple factors. These include mechanism of injury, primary injury location, time to thoracotomy, and various physiologic predictors such as prehospital CPR, signs of life, and cardiac rhythm. A review of over 10,000 patients who underwent resuscitative thoracotomy reported an overall rate of survival to hospital discharge of 8.5% and an overall rate of intact neurologic status of 85% for survivors. Survival following resuscitative thoracotomy for blunt trauma is 2.3% compared to 10.6% for penetrating injury. Survival following



resuscitative thoracotomy for stab wounds is 15.8% compared to 7.2% for gunshot wounds. Cardiac injuries carry the best prognosis, with 17.3% of patients surviving resuscitative thoracotomy, compared to 10.5% for thoracic noncardiac wounds and 7% for abdominal, neck, or extremity wounds. Finally, for those patients in whom signs of life or vital signs are obtained in the emergency department, 19% and 17.4%, respectively, survive resuscitative thoracotomy. This is in comparison to those patients without prethoracotomy signs of life or vital signs in the emergency department, in whom only 2.9% and 3.8%, respectively, survive.

Successful resuscitative thoracotomy requires choreographed resuscitation of the patient with many simultaneous actions occurring in parallel. For example, all patients undergoing resuscitative thoracotomy require a definitive airway, right-sided chest tube or finger thoracostomy, and adequate vascular access with blood products available for hemodynamic support. Prior to beginning resuscitative thoracotomy, one must also consider the availability of those resources necessary to provide definitive care for these gravely injured patients once the thoracotomy is complete. Each trauma center should have policies in place regarding the multidisciplinary approach to resuscitative thoracotomy, recognizing that resuscitative thoracotomy is not a location-specific procedure and may be considered in any setting where the procedural means and postprocedural care systems are available. Finally, throughout this high-intensity procedure, coordination is of the utmost importance to ensure the safety of the entire treatment team as the procedure places the performing providers at high risk for blood-borne pathogen exposure. Despite recognizing the reality of these complex issues, the most common pitfall encountered during resuscitative thoracotomy is the cognitive error of hesitating to consider this potentially lifesaving procedure.

Available equipment, including a thoracotomy tray, 3-0 polypropylene monofilament or silk suture, a suction device, internal defibrillator paddles, and ACLS medications, should be prepared. In situations where this equipment is unavailable, the minimal equipment necessary to complete the procedure is a scalpel, scissors, and rib spreader. The patient should be supine with the upper extremities abducted. The bilateral thoraces should be rapidly prepped with an iodine bath and draped while the operator and assistant don surgical masks with eye shield, sterile gowns, and double-layered gloves.

The initial approach is a left anterolateral thoracotomy. The incision should be made in the fourth intercostal space, which often corresponds with the nipple in a male and with the inframammary crease of the superiorly retracted breast in a female. The incision is made with a number-10 or number-20 scalpel, beginning at the sternal border and curving along the path

of the rib, extending to the midaxillary line. Sharp dissection with one to two firm strokes of the scalpel carries the incision down to the intercostal muscles. Upon reaching this level, the scalpel is set aside and Mayo scissors are used to incise the intercostal muscles and the visceral pleura along the length of the incision. This incision is made above the rib to avoid damaging the intercostal neurovascular bundle. Just prior to incising the pleura, pause ventilations to allow the left lung to collapse away from the chest wall. Next, insert a Finochietto retractor (rib spreader) between the ribs with the handle and ratchet bar oriented toward the floor so as to allow the left anterolateral thoracotomy incision to be carried across to the right chest in a clamshell fashion if there is evidence of injury to the right hemithorax or the right chest tube output is significant.

Upon entry to the thoracic cavity, any uncontained blood in the chest must be evacuated promptly to allow for visualization of vital structures. The left lung should be mobilized by transecting the inferior pulmonary ligament. If catastrophic pulmonary hemorrhage is encountered, one may consider cross-clamping the pulmonary hilum or rotating the lung 180 degrees about the hilum to staunch the pulmonary blood supply. Next, with the lung displaced, grasp and tent the pericardium with tissue forceps and use Metzenbaum scissors to incise the pericardium in a linear fashion parallel and anterior to the left phrenic nerve. The operator's gloved finger can also be used to bluntly open the pericardium. With the pericardium opened, evacuate any blood products contributing to tamponade physiology and deliver the heart through the pericardiotomy into the left thoracic cavity to begin two-handed open cardiac massage, placing the compressor's fingers in a position that avoids constricting the coronary arteries. Cardiac injuries should be occluded with digital pressure or temporarily closed with staples. The operator may also consider inserting a Foley catheter into a cardiac wound that is too large to control with direct pressure. Once the catheter is inserted, the balloon is inflated to temporarily tamponade bleeding prior to definitive repair. Given the technical difficulty of cardiorrhaphy and the risk of causing further damage to the myocardium, when possible, the emergency physician should defer definitive repair of cardiac wounds to the trauma or cardiac surgeon. When unavoidable, the emergency physician may place horizontal mattress 2-0 or 3-0 polypropylene monofilament or silk sutures with pledgets to repair cardiac wounds.

Next, attention is turned to cross-clamping the descending thoracic aorta to improve cerebral and coronary perfusion in the setting of persistent hypotension. After superiomedially retracting the left lung, advance the hand along the posterior thoracic wall toward the spinal column, bluntly open the mediastinal pleura, and bluntly dissect the aorta away from the esophagus.

Identifying the aorta may be challenging as the esophagus can be mistaken for the aorta. Anatomically, the aorta is the structure immediately anterior to the spine, and placement of a gum elastic bougie or an orogastric tube into the esophagus helps differentiate these two structures. Once the aorta is identified and isolated from the esophagus, flex the left index finger around the vessel and place an aortic or DeBakey vascular clamp to occlude distal blood flow. Potential complications of resuscitative thoracotomy that should be minded include phrenic nerve injury, coronary artery injury, postprocedure infection, and injury or disease transmission to health care workers.

Patients who regain vital signs following initial resuscitative efforts and completion of the resuscitative thoracotomy must be transferred immediately to the operating room for definitive management of their injuries.

While the resuscitative thoracotomy has been practiced for over half a century, studies of the utility of novel techniques in the management of cardiopulmonary arrest secondary to trauma are ongoing. For example, case series of EMS physician-staffed prehospital systems have reported higher survival rates when resuscitative thoracotomy is completed in the prehospital environment. Furthermore, research into new tools—such as resuscitative endovascular balloon occlusion of the aorta—is ongoing and may augment or replace the resuscitative thoracotomy in the future.

## KEY POINTS

- Resuscitative thoracotomy is indicated for penetrating thoracic injury with observed signs of life prior to cardiopulmonary arrest.
- Resuscitative thoracotomy may be considered for penetrating nonthoracic injury and blunt traumatic injury, though rates of survival are low.
- Survival and neurologic recovery following resuscitative thoracotomy is highest for patients with witnessed signs of life, penetrating trauma, stab wounds, and cardiac injury. Correct placement of the rib spreader with the handle and ratchet bar oriented toward the floor is necessary to prevent interfering with the ability to extend the left anterolateral thoracotomy incision to a clamshell incision.
- Resuscitative thoracotomy should be undertaken only if the treating facility has resources in place to provide definitive care for the patient and if the safety of all treatment team members can be assured.

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## INCREASED ICP IN RESUSCITATION

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The skull is a fixed compartment that holds three main components: blood, cerebrospinal fluid (CSF), and brain parenchyma. When the volume of one component increases, the volume of the others must decrease in order to maintain a constant pressure. Cerebral autoregulation ensures this mechanism of balance and compensation. When a change in volume is too extreme, or occurs too rapidly, cerebral autoregulation mechanisms can be overwhelmed and may be unable to compensate, resulting in increased intracranial pressure (ICP).

Elevated ICP is harmful in that it can compromise cerebral perfusion pressure (CPP). CPP can be calculated by subtracting ICP from mean arterial pressure (MAP). In other words,  $CPP = MAP - ICP$ . When CPP is lowered, this leads to decreased brain tissue perfusion and oxygenation. Additionally, extreme elevations in ICP may cause brain herniation down the pathway of least resistance—the foramen magnum, resulting in compression and anoxia of the brainstem, resulting in death.

Generally recognized guidelines are a target CPP of  $>60$  mm Hg and ICP  $< 20$  mm Hg. In reality, emergency medicine clinicians will not have the luxury of knowing the patient's exact ICP (measured via ICP monitor). Suspicion for elevated ICP must be inferred based on signs, symptoms, and mechanism of injury. Look for changes in mental status ranging from agitation to lethargy, a focal neurologic deficit, anisocoria, nonreactive pupils, or the classic Cushing triad of bradycardia, hypertension, and irregular respirations.

Suspicion for elevated ICP should be high in trauma patients (because of hemorrhages or cerebral edema), hypertensive patients with mental status change or severe headache (intracranial hemorrhage), and anyone on anticoagulants with a change in mental status, headache, or focal neurologic

deficit. Patients with large volume ischemic strokes can have elevated ICP because infarcted brain tissue may become edematous. Patients with brain tumors can deteriorate due to an acute increase in vasogenic edema. Acute hydrocephalus can present in patients with shunt failure and in patients with subarachnoid hemorrhage (SAH).

When you suspect elevated ICP, address the airway, breathing, and circulation (ABC) first. Address hypoxia and hypotension, which are both detrimental to brain tissue. Intubate early if you anticipate deterioration in clinical status. When intubation is delayed, the patient may worsen and start to retain CO<sub>2</sub>, leading to cerebral vasodilatation. This will further increase ICP in the brain-injured patient. Consider neurosurgical consult, as many of these patients will need operative intervention or ICP monitors. Check a coagulation profile, and fix whichever coagulopathies you can, bearing in mind that many modern anticoagulants cannot be monitored by blood levels and can be difficult to reverse. Elevate the head of the bed to 30 to 45 degrees. Keep the neck straight. Fever, pain, agitation, and discomfort due to the ventilator all increase ICP, so treat these problems aggressively. You can hyperventilate the patient (to a PaCO<sub>2</sub> of 30 to 35 mm Hg) but only as a temporizing measure. It should not be continued for more than an hour.

Hypertonic therapy is the staple of medical management of intracranial hypertension. This can be achieved in a number of ways. *Mannitol* is an osmotic diuretic. It draws free water out of the brain parenchyma into the blood vessels (lowering ICP) and then acts as a diuretic.

Dosing for acute reduction in ICP is 1 g/kg of a 20% solution given over 20 minutes, but may be given faster in a dire situation. It can be given through peripheral or central venous access. Mannitol often takes 5 to 10 minutes to draw up because it crystallizes. So give your nurses a heads up if you think you're going to need it.

Prior to administration of mannitol, baseline sodium and serum osmolality level should be obtained. Further therapy will be guided by serially checking these values. Anticipate very high urine outputs and place a Foley catheter. This will prevent urinary retention, which can further increase their ICP. Do not give mannitol to hypotensive patients as it frequently causes a mild drop in blood pressure. Remember to administer maintenance fluids if giving mannitol long term as it can cause dehydration. Avoid using mannitol in patients with renal failure, as it can worsen renal function.

One possible adverse effect of mannitol is rebound intracranial hypertension, which can occur as its effects are wearing off or when repeat dosing is stopped. This occurs when fluid shifts from the brain vasculature back into the brain tissue.

*Hypertonic saline* works via a very similar mechanism to mannitol, and its onset is about as rapid at 5 to 10 minutes. It does not act as robustly as a diuretic, making it a better plasma expander. Because of this, it is a more desirable choice in patients in whom hypotension or hypovolemia is a problem and your goal is to increase cardiac output. It is also preferred over mannitol in patients with renal failure. Hypertonic saline is believed to not have the risk of rebound intracranial hypertension when compared to mannitol. Conversely, caution must be exercised in patients who are fluid overloaded.

The major downside to hypertonic saline is that most institutions require it to be given via central venous access due to potential blood vessel and tissue injury. In the hyperacute setting, the risks and benefits must be weighed. In certain situations, 3% saline may be started peripherally while central access is being established. Alternatively, 2% saline solutions can be administered peripherally.

The dosing regimens for hypertonic saline are not as well studied as mannitol. For acutely lowering ICP, it is common practice to use a bolus of 250 to 500 mL of 3% saline, or 30 mL of 23.4% saline. As with mannitol, baseline sodium and serum osmolality levels should be obtained prior to administration.

## KEY POINTS

- Treat the whole patient first; don't forget your ABC's.
- Hypoxia, hypotension, and hypercarbia must be avoided in the setting of elevated ICP.
- Hypertonic saline is superior to mannitol in the setting of hypotension or hypovolemia.
- Know the dosing and administration guidelines for mannitol and hypertonic saline and don't be stingy with it if your patient really needs it.

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# MASSIVE PULMONARY THROMBOEMBOLISM AND THROMBOLYTICS

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NOUR AL JALBOUT, MD

Venous thromboembolism (VTE), which includes deep vein thrombosis (DVT) and pulmonary embolism (PE), is responsible for more than 250,000 annual hospitalizations in the United States, with significant risk for morbidity and mortality. The most serious clinical presentation of VTE is acute PE.

Risk factors for VTE must be considered as they may help the provider zero in on the diagnosis of PE. Extrinsic risk factors include surgery, trauma, immobilization, and oral contraceptive or hormonal replacement therapy use. Intrinsic risk factors are mainly due to hypercoagulable states, including pregnancy, malignancy, and various coagulation disorders. Lastly, VTE may occur idiopathically. The clinical symptoms of PE may include chest pain, dyspnea, syncope, and symptoms of DVT. Objective signs of PE include tachycardia, hypotension, hypoxia, tachypnea, fever, altered mental status, and signs of DVT (e.g., asymmetric extremity edema, or positive Homans sign). One of the most life-threatening presentations of PE is massive PE. The American Heart Association (AHA), in its 2011 scientific statement, defined massive PE as:

*Acute PE with sustained hypotension (systolic blood pressure 90 mm Hg for at least 15 minutes or requiring inotropic support, not due to a cause other than PE, such as arrhythmia, hypovolemia, sepsis, or left ventricular [LV] dysfunction), **pulselessness**, or **persistent profound***

***bradycardia*** (heart rate < 40 beats per minute with signs or symptoms of shock).

The differential diagnosis for a patient presenting with symptoms of massive PE includes acute coronary syndrome, cardiac tamponade, valvular dysfunction, acute pulmonary edema, pneumothorax, and aortic dissection. When presented with a patient in whom these diagnoses are being considered, bedside transthoracic echocardiography is extremely useful. In this scenario, it may not be feasible to transport an unstable patient to have radiographic studies. Ultrasonographic findings associated with massive PE include dilation of the right ventricular (RV) or inferior vena cava (IVC) RV dysfunction, septal flattening, and pulmonary hypertension. The clinician may also look for signs of DVT using lower extremity Doppler. Other findings such as cardiac tamponade, pneumothorax, and large pleural effusion may also be confirmed via bedside ultrasound, helping the provider key in on a diagnosis.

How does PE compromise circulation and eventually lead to shock? Acute PE interferes with the circulation in the pulmonary bed. If more than 30% to 50% of the total cross-sectional area of the pulmonary arterial bed is occluded by thromboemboli, the pulmonary artery pressure (PAP) increases. Additionally, local hypoxia induces vasoconstriction in the vascular bed, leading to a further increase in the pulmonary vascular resistance. As such, the abrupt increase in pulmonary vascular resistance results in RV dilation, with resultant increase in RV pressure and volume. The sudden increase in wall tension prevents the RV from overcoming the PAP, leading to leftward bowing of the interventricular septum. This impedes LV filling in early diastole, eventually reducing the cardiac output and contributing to hemodynamic instability and obstructive shock.

Patients presenting with symptoms of massive PE must have their airway, breathing, and circulation immediately addressed. As massive PE causes obstructive shock, treatment is required to restore adequate circulation. Per the AHA 2011 scientific statement, fibrinolysis is reasonable for patients with massive acute PE who have acceptable risks of bleeding complications (Class IIa; Level of Evidence B). The data are derived from 13 main randomized controlled trials studying thrombolytics compared to placebo. Only a small subset included an investigation of massive PE, with primary outcomes being variable and conflicting in terms of trends toward improved survival and decrease in recurrent PEs. The main positive outcomes observed with thrombolytics were stabilization of respiratory and cardiovascular function without vasopressor support, reduction of RV damage, and prevention of PE recurrence, with an increased probability of

survival. The main harms inflicted were major hemorrhage, including intracerebral hemorrhage, and increased risks of minor hemorrhages, resulting in prolonged length of stay and potential blood product administration. The decision to administer a fibrinolytic agent depends on an individualized assessment of the risks and benefits. The optimal medical decision must incorporate patient wishes, life expectancy, and his/her risk of bleeding complications.

Once the decision to administer thrombolytic therapy has been made, the thrombolytic agent is typically administered via a peripheral intravenous catheter as an infusion. The FDA-recommended infusion dose is for alteplase at 100 mg as a continuous infusion over 2 hours while withholding anticoagulation during the 2-hour infusion period. For this reason, when massive PE is suspected, anticoagulants with prolonged half-lives (such as low molecular weight heparin) should be avoided. Anticoagulation is typically started after clot lysis with heparin and warfarin. The optimal duration of anticoagulation is based upon extrapolated evidence from patients with acute DVT in whom thrombolysis was not performed, which is a minimum of 3 months. Patients should be sent to an intensive care unit for further monitoring and treatment.

If patients survive this acute event, the main emphasis then turns to rehabilitation, anticoagulation compliance, and ultimate prevention of any predisposing factors for future VTE. Although further clinical trials of the advanced therapies for VTE are needed, your decision must be based on the best available evidence. The administration of thrombolytics may lead improved morbidity and mortality and should be considered in the appropriate clinical scenario.

## KEY POINTS

- Clinical decision rules such as Wells' criteria, Geneva score, and PERC (PE rule out criteria) are available to help determine pretest probability of VTE.
- Unexplained shock may be obstructive shock secondary to PE.
- Bedside ultrasound is a valuable tool when considering massive PE in a patient.
- Weigh risks and benefits when considering thrombolytic administration, paying special attention to bleeding risks.
- Emphasize compliance with anticoagulation after disposition.

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# FLUID IN THE SAC? CARDIAC TAMPONADE

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NGOZI NWEZE, MD

## INTRODUCTION

The pericardial sac is an elastic membrane that surrounds the pericardium. Pericardial effusion is caused by fluid (blood, pus, serous, neoplastic) accumulating within the pericardial sac. As fluid accumulates and the pericardium is maximally stretched, it eventually leads to compression of the atria and ventricles, decreased venous return and, subsequently, decreased cardiac output. Gradual accumulation of fluid allows the pericardium to accommodate and stretch over time, but rapid accumulation such as in ventricular free wall rupture or penetrating trauma does not allow time for the pericardial sac to stretch. Cardiac tamponade occurs when cardiac output is significantly decreased and compensatory mechanisms are no longer adequate, leading to hemodynamic compromise and collapse.

## CLINICAL PRESENTATION AND PHYSICAL EXAM

Patients can present with dyspnea, palpitations, pleuritic chest pain, and lethargy. Physical exam may reveal pulsus paradoxus (decreased blood pressure during inspiration as increased intrathoracic pressure reduces venous return), tachycardia, tachypnea, muffled heart sounds, jugular venous distention, and hypotension (the last three signs making up Beck triad).

## DIAGNOSIS

Electrocardiogram (EKG) may reveal electrical alternans (alternation of the

height of QRS complexes as the heart “swings” within the fluid-filled pericardium), low QRS voltage, sinus tachycardia, arrhythmia, or nonspecific ST changes. Chest radiograph may reveal enlarged cardiac silhouette. Bedside echocardiography may reveal right ventricular collapse during diastole, leftward septal deviation during inspiration, inferior vena cava collapse during inspiration, right atrial collapse during systole, or swinging of the heart within the pericardial sac.

## MANAGEMENT

Intravenous fluid bolus should be given judiciously in an attempt to increase venous return, as too much fluid can worsen tamponade. The use of inotropes is controversial as endogenous catecholamines are already working to increase inotropy of the heart. If inotropes must be used, dobutamine is preferred. The definitive management involves needle pericardiocentesis, preferably under ultrasound guidance. After cleansing and anesthetizing the insertion site, a syringe and a needle that is at least 1.5 inches long is used to pierce the skin in the subxiphoid region at a 45-degree angle, directed toward the left shoulder. Applying continuous negative pressure, enter the pericardial sac (preferably under ultrasound guidance). Fluid should be aspirated until hemodynamic status is improved. Contraindications to pericardiocentesis include traumatic hemopericardium, myocardial free wall rupture, and aortic dissection. Surgical management is definitive in these situations. Complications of pericardiocentesis include infection, injury to the liver and other intra-abdominal structures, pneumothorax, myocardial laceration, intercostal or internal mammary vessel laceration or aneurysm, pericardial thrombus, false-positive aspiration (intracardiac aspiration of blood), false-negative aspiration (due to clotted blood), dysrhythmia, and cardiac arrest.

### KEY POINTS

- Consider cardiac tamponade in hypotensive patients. There are numerous conditions that can lead to tamponade.
- Bedside ultrasound is usually diagnostic.
- Use intravenous fluids judiciously as fluid overload can worsen status.
- Ultrasound-guided pericardiocentesis is the definitive management in tamponade except in the setting of trauma, ventricular free wall rupture, or aortic dissection.

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# IS IT WIDE OR IS IT NARROW? PEA: A SIMPLIFIED APPROACH TO PULSELESS ELECTRICAL ACTIVITY

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**NICHOLAS RISKO, MD, MHS**

Pulseless electrical activity (PEA) arrest occurs when a patient has organized electrical activity on electrocardiogram (ECG) but no pulse. PEA is identified in ~20% to 40% of cardiac arrest patients, and overall survival is poor compared to ventricular tachycardia or ventricular fibrillation rhythms. Studies have shown that roughly 40% of these patients have some mechanical cardiac activity; however, it is not sufficient to generate a palpable pulse.

The PEA arrest has always been a management challenge, as a physician is burdened with recalling long lists of causes that require skillful and rapid intervention. In this chapter, we suggest a simplified approach to PEA arrest, building on recent peer-reviewed literature.

The common training requires memorization of ten to thirteen “Hs and Ts” (depending how you count and whether you are referencing the American Heart Association or European Society of Cardiology guidelines) and to calmly recall them during the chaotic environment of an arrest. Hs include hypovolemia, hypoxia, hydrogen ion (acidosis), hypo- or hyperkalemia, hypoglycemia, hypocalcemia, and hypothermia. Ts include tension pneumothorax, tamponade, toxins, thrombosis (pulmonary or cardiac), and trauma. While emergency physicians rise to the task on a daily basis, this error-prone system requires constant practice and preparation. Furthermore, many of these etiologies are theoretical with a negligible or unproven contribution to the burden of PEA arrest. For example, reviewers



have had difficulty finding evidence that hypokalemia, hypoglycemia, and hypothermia have ever caused a PEA arrest. Meanwhile, the prevalence of pulmonary embolism as a cause of PEA ranged from 36% to 68% in several small case series.

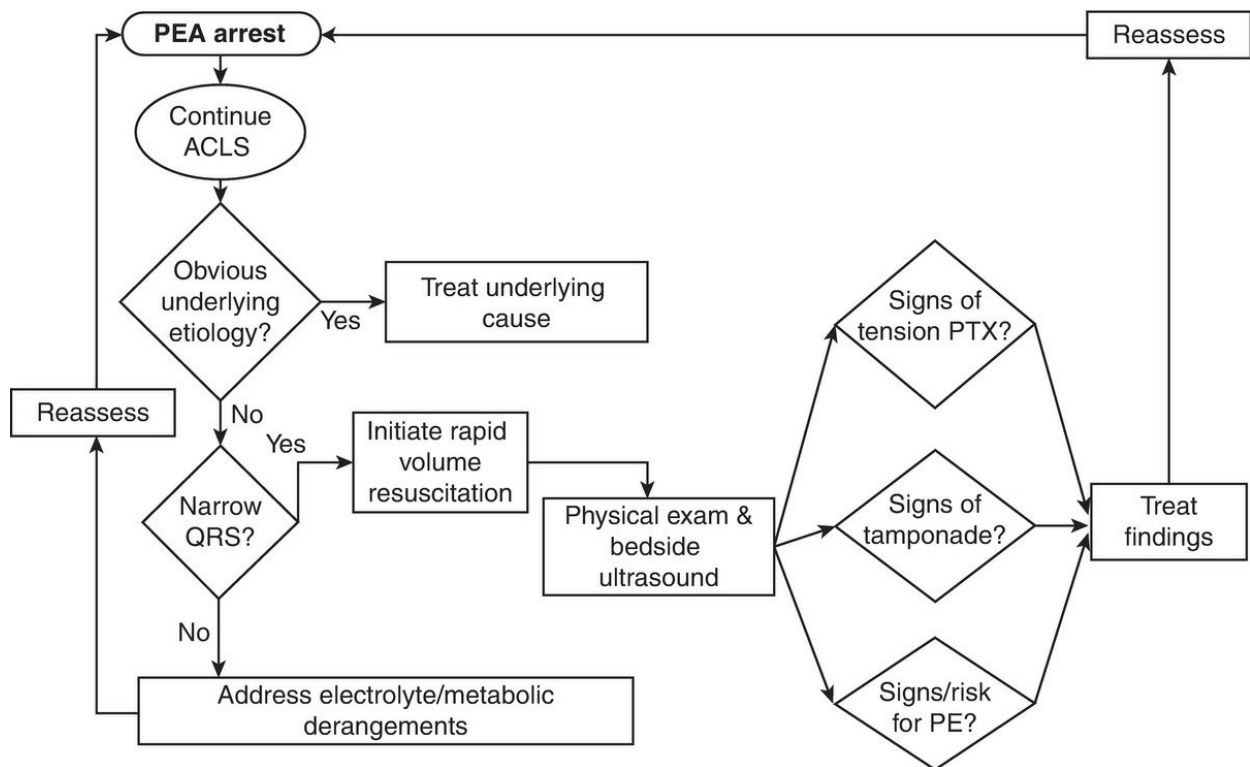
Desbiens proposes a simplified approach to PEA management named the “3 and 3 rule.” He asks you to consider the three most likely causes of PEA: severe hypovolemia, obstruction to circulation, and pump failure. Finally, for obstruction to circulation, he highlights the three principal causes: tamponade, tension pneumothorax, and pulmonary embolism. He suggests using the absence of a femoral pulse during CPR to imply obstruction or hypovolemia, and its presence to imply other causes.

Another simplified approach involves ECG. Electrolyte derangements that may lead to PEA often have characteristic ECG findings, such as J waves, T-wave changes, or QT changes. Furthermore, rapid narrow complex rhythms are likely to represent a physiologic response to circulatory collapse in the otherwise healthy heart, while widening of the QRS may represent decline to the point of intrinsic cardiac failure or severe metabolic derangement. It has been noted that widening of the QRS, lengthening QT, slowing rate, and lack of atrial activity all portend poorer prognosis.

Littman et al. propose a novel algorithm based upon ECG evaluation of the QRS as narrow ( $<0.12$  s) or wide ( $>0.12$  s) and focusing only on most likely etiologies in the nontrauma setting. Narrow QRS PEA means a mechanical problem such as tamponade, tension pneumothorax, pulmonary embolism, mechanical hyperinflation, or myocardial rupture. Wide QRS means metabolic problems such as severe hyperkalemia, toxins, or ischemia. The authors suggest initiating IV fluids for the narrow complex group before contemplating further therapies. For wide complex, they suggest empiric IV calcium chloride (for hyperkalemia) and sodium bicarbonate (for potential sodium blocker toxicity or reduced transport secondary to ischemia).

Bedside ultrasound is another helpful adjunct. Hernandez et al. present a sonographic algorithm for cardiac arrest utilizing techniques familiar to emergency physicians to rapidly assess for hypovolemia, tension pneumothorax, cardiac tamponade, pulmonary embolus, and gross ventricular function. [Figure 28.1](#) presents a suggested algorithm that builds from the work above.

Remember that as hypoperfusion progresses, an initially mechanical arrest may degrade into a metabolic one. PEA is an evolving continuum that requires adaptive and agile management. We hope this chapter has stimulated some critical thought on how to approach your next PEA arrest.



**Figure 28.1** Suggested pathway for the management of PEA arrest.

## KEY POINTS

- There is a lack of evidence to justify many of the “Hs and Ts” as causes of PEA arrest.
- Pulmonary embolus is a common cause of cryptogenic PEA arrest.
- In addition to history and physical exam, use ECG and ultrasound to help identify etiology.
- Narrow QRS means better prognosis and a mechanical etiology with the potential for reversal after rapid and appropriate intervention.
- Utilize this chapter and the readings below to develop an approach to PEA arrest that works well for you, is easy to remember, and is high yield for reversible arrests.

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## UNDIFFERENTIATED SHOCK

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**DANIEL SHEETS, MD, MPH AND RANDALL T. RHYNE,  
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The patient in undifferentiated shock presents a unique challenge for the emergency physician, as the provider must perform time-critical resuscitation with limited diagnostic information. In addition to a focused assessment of the ABC's during resuscitation, special attention should be paid to "the pump," "the tank," and "the pipes" to provide a systematic approach to the hypotensive patient. Early recognition of the etiology of hypotension and timely intervention can prevent significant morbidity and mortality, as untreated shock is almost always a fatal diagnosis.

Shock is broadly defined as an imbalance in tissue oxygen demand and tissue oxygen supply. This imbalance results in cellular injury with toxic increases in intracellular calcium, anaerobic respiration, lactate production, cellular death, and the release of systemic proinflammatory cytokines. This cascade ultimately results in end-organ damage and may progress to multiple organ dysfunction syndrome. The four broad classifications of shock are **hypovolemic**, **cardiogenic**, **obstructive**, and **distributive**. Hypovolemic shock may be due to hemorrhage in the setting of trauma or due to fluid losses from severe vomiting or diarrhea. Cardiogenic shock results from decreased cardiac output due to an intrinsic heart defect, often from myocardial infarction, valvular rupture, or congestive heart failure. Obstructive shock results from a physical obstruction that reduces cardiac output, and may occur due to cardiac tamponade, massive pulmonary embolism, or tension pneumothorax. Distributive shock is most commonly due to sepsis and overwhelming infection, though it may also be caused by anaphylaxis or neurogenic shock in spinal cord injury. The early identification of the type of shock can often be elicited through patient history, physical exam, laboratory investigations, ECG findings, chest x-ray,

and bedside ultrasound.

The initial step in the resuscitation of the patient in undifferentiated shock includes obtaining peripheral venous access and ensuring adequate ventilation and oxygenation. This may involve endotracheal intubation, as the failure of mask ventilation or positive pressure ventilation in the hypotensive patient may result in respiratory failure and circulatory collapse. A trial bolus of crystalloid solution should be considered, as even those patients in acute cardiogenic shock may be intravascularly depleted. Careful monitoring of clinical response to fluid should be maintained, as pulmonary edema can be an unintended consequence of fluid administration.

An early focus on “the pump” helps discriminate between the four broad classifications of shock and decreases time to lifesaving interventions. An ECG should be performed early to assess for STEMI as a cause of cardiogenic shock that necessitates immediate percutaneous coronary intervention (PCI). ECG may also demonstrate right heart strain suggesting massive pulmonary embolism or electrical alternans suggesting cardiac tamponade. Bedside cardiac ultrasound quickly demonstrates the presence of pericardial effusion and provides an assessment of cardiac contractility. The presence of a large pericardial effusion with right ventricular collapse suggests cardiac tamponade causing obstructive shock, and immediate pericardiocentesis is indicated to aid diastolic filling. An acutely dilated right ventricle may suggest massive pulmonary embolism causing right heart strain and obstructive shock. A hyperdynamic heart with normal or high contractility suggests distributive or hypovolemic shock. A hypodynamic heart with decreased contractility and dilated cardiac chambers suggests cardiogenic shock.

Following evaluation of “the pump,” an assessment of “the tank” helps determine intravascular volume status and fluid responsiveness. Physical exam findings may provide clues as to whether “the tank” is empty or full. Elevated jugular venous pressure, bilateral rales on lung auscultation, or lower extremity edema suggest fluid overload but may not be entirely representative of intravascular volume. A passive leg raise may be performed to temporarily increase venous return and determine fluid responsiveness. Bedside ultrasound provides valuable clues regarding a patient’s intravascular status. An inferior vena cava width  $< 2$  cm, as measured 2 to 3 cm distal from the right atrial junction, with inspiratory collapse of  $>50\%$  suggests the patient is intravascularly depleted and may benefit from intravenous fluids. This finding is also suggestive of hypovolemic or distributive shock. As a caveat, IVC evaluation must be performed prior to intubation as positive pressure ventilation makes IVC assessment unreliable. Further evaluation of “the tank” for intraperitoneal free fluid and pleural

fluid may suggest hemorrhage as a cause of hypovolemic shock. Ultrasound may be used to reassess intravascular status after fluid resuscitation. Vasopressors may be necessary should a patient continue to be hypotensive despite adequate fluid repletion.

Finally, evaluation of “the pipes” should be considered in the patient with undifferentiated shock. Physical exam may reveal a pulsatile abdominal mass suggestive of aortic aneurysm, though this finding is not sensitive. Bedside ultrasound may be used to evaluate the abdominal aorta for aneurysm or dissection, and in the setting of hypotension a ruptured AAA must be considered. Ultrasound of “the pipes” of the lower extremities may be performed to assess for deep vein thrombosis, which may suggest massive pulmonary embolism and obstructive shock.

A systematic evaluation of “the pump,” “the tank,” and “the pipes” can aid in the prompt recognition of the etiology of undifferentiated shock and help guide therapy (see [Table 29.1](#)). Early recognition of shock classification and prompt intervention can save lives.

**TABLE 29.1 ULTRASONOGRAPHIC FINDINGS AND CORRESPONDING INTERVENTIONS FOR THE FOUR BROAD CLASSIFICATIONS OF SHOCK**

| <b>CLASSIFICATION OF SHOCK</b> | <b>“THE PUMP”</b>  | <b>“THE TANK”</b>                          | <b>“THE PIPES”</b>       | <b>INTERVENTION</b>   |
|--------------------------------|--|--|--------------------------|---|
| Hypovolemic                    | Normal or high contractility<br>Hyperdynamic<br>Small cardiac chambers   | Thin (<2 cm) IVC<br>>50% collapsibility    | AAA<br>Aortic dissection | IV fluid  |
| Distributive                   | Normal contractility<br>Hyper-dynamic<br>Normal cardiac chambers   | Thin (<2 cm) IVC<br>>50% collapsibility    | Normal                   | IV fluid<br>Vasopressors<br>Antibiotics<br>Anaphylaxis Tx   |
| Cardiogenic                    | Decreased contractility<br>Hypodynamic<br>Large cardiac chambers   | Dilated (>2 cm) IVC<br><50% collapsibility | Normal                   | Vasopressors<br>Inotropes   |
| Obstructive                    | Tamponade: pericardial effusion, small cardiac chambers<br>PE: dilated right ventricle, small left ventricle<br>Tension PTX: small cardiac chambers, absence of lung sliding | Dilated (>2 cm) IVC<br><50% collapsibility | Lower extremity DVT      | Tamponade: pericardiocentesis<br>PE: thrombectomy or thrombolytics<br>Tension PTX: needle compression |

## KEY POINTS

- Remember the four classifications of shock: hypovolemic, distributive, cardiogenic, and obstructive.
- Use a systematic approach to “the pump,” “the tank,” and “the pipes” to determine classification of shock.
- Integrate the use of bedside ultrasound into resuscitation of patients with undifferentiated shock to help make the diagnosis and guide interventions.

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# **KNOW HOW TO IDENTIFY ABDOMINAL COMPARTMENT SYNDROME**

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## **INTRODUCTION**

Current diagnosis and treatment for abdominal compartment syndrome (ACS) is driven by expert panel consensus guidelines. The most widely recognized source is the World Society of the Abdominal Compartment Syndrome (WSACS), which released its updated guideline in 2013. ACS occurs when elevated intra-abdominal pressure (IAP), or, intra-abdominal hypertension (IAH), leads to new organ dysfunction. The incidence of ACS in the ED has not been reported, but in studies of trauma patients, the incidence is between 1% and 14%.

## **DIAGNOSIS**

Clinical signs of ACS can be broken down by organ system. In the brain, IAH impairs venous return, which increases intracerebral pressure and decreases cerebral perfusion pressure. In the heart, IAH increases intrathoracic pressure, which decreases venous return and cardiac output. In the lungs, IAH pushes up on the diaphragm, which decreases vital capacity and lung compliance leading to hypoxemia and hypercarbia. In the GI tract, IAH decreases abdominal perfusion pressure (APP), which is the mean arterial pressure minus IAP. Lower APP leads to mesentery hypoperfusion

and poor lactate clearance. In the kidneys, IAH decreases renal blood flow, which manifests as acute kidney injury and oliguria.

Risk factors for ACS can be grouped by mechanism. Etiology of ACS is divided into primary and secondary causes. Primary causes include bowel obstruction, ascites, pancreatitis, trauma, recent major abdominal surgery, and ruptured abdominal aortic aneurysm. These conditions often require early surgical or interventional radiology therapies. Secondary causes of ACS are from nonabdomen/pelvis pathology and include massive fluid resuscitation/transfusion, shock, and severe burns.

Physical exam, clinical signs, and imaging are not reliable in identifying ACS. Upon evaluation in an emergency setting, if a patient's history contains any of the risk factors in [Table 30.1](#), it is reasonable to consider ACS in the differential diagnosis. Key exam and lab findings may include abdominal distention, progressive oliguria, hypercarbia, refractory hypoxemia, and lactic acidosis.

**TABLE 30.1 ABDOMINAL COMPARTMENT SYNDROME RISK FACTORS**

|                                      |   |
|--------------------------------------|---|
| Diminished abdominal wall compliance | Abdominal surgery<br>Major trauma/burns<br>Prone positioning  |
| Increased intraluminal contents      | Gastroparesis/distention<br>Ileus<br>Colonic pseudo-obstruction<br>Volvulus   |
| Increased abdominal contents         | Acute pancreatitis<br>Ascites<br>Hemo-/pneumoperitoneum<br>Intra-abdominal infection/tumors<br>Peritoneal dialysis  |
| Capillary leak                       | Acidosis<br>Damage control laparotomy<br>Hypothermia<br>Massive fluid resuscitation >5 L/24 h<br>Polytransfusion >10 units/24 h   |
| Other                                | Age<br>Sepsis<br>Coagulopathy<br>Increased head of bed angle (>20 degrees)<br>Massive incisional hernia repair<br>Obesity or increased body mass index<br>Mechanical ventilation<br>PEEP > 10 |

Ultimately, to objectively evaluate for IAH/ACS, it is necessary to measure IAP using transduced intravesicular pressures ([Table 30.2](#)). Peritoneal adhesions, pelvic hematomas/fractures, abdominal packing, or a neurogenic bladder may affect the accuracy of IAP measurement. Patients with morbid obesity, pregnancy, or long-standing ascites could have chronically elevated IAP. Per WSACS definitions, IAP is the steady-state pressure within the abdominal cavity expressed in mm Hg and measured at end-expiration in the supine position. Elevated IAP is considered to be IAH when it is  $\geq 12$  mm Hg. Normal IAP for healthy adults is 0 to 5 mm Hg. Normal IAP for critically ill adults is 5 to 7 mm Hg. For adults, ACS occurs when IAP is maintained above 20 mm Hg (for pediatrics, the threshold is 10 mm Hg) with new organ dysfunction. The diagnosis of ACS is not dependent upon APP values.

**TABLE 30.2 SAMPLE SETUP AND STEPS TO MEASURE ABDOMINAL COMPARTMENT PRESSURE**

1. Clamp the drainage tube of the Foley catheter.
2. Instill up to 25 mL sterile saline into the bladder via the aspiration port of the Foley catheter. Be certain the catheter is filled with saline!
3. Attach a pressure transducer to an 18-gauge needle and insert into the aspiration port.
4. Zero the transducer at the level of the mid-axillary line.
5. With the patient in the supine position, ensure that abdominal muscle contractions are absent and measure the bladder pressure at end-expiration.

Reproduced from Gestring M. Abdominal compartment syndrome. *UpToDate*. 2015.

## TREATMENT

Early recognition of ACS is crucial given the reported association with ~40% to 100% mortality. Several temporizing medical therapies can be implemented based on the suspected underlying mechanism for ACS. For abdominal wall compliance, appropriate pain and sedation meds should be used; trials of neuromuscular blockade to reduce muscle tone can be attempted and, if the patient's cardiovascular and respiratory status allow for it, supine positioning. For increased intraluminal contents, nasogastric and rectal decompression may help lower IAP. For increased intra-abdominal contents such as ascites, hematoma, or abscess, ultrasound-guided

paracentesis in the ED or percutaneous drainage by interventional radiology should be considered, respectively. At this time, WSACS has no recommendations regarding diuretics or albumin usage.

In an emergency setting, if there is concern for ACS, first obtain an IAP. If it is above 12, begin appropriate medical therapies to lower IAP, obtain an early surgical consult, and measure IAP every 4 hours. If the IAP does not drop or organ dysfunction progresses, medical management is considered unsuccessful and surgical intervention is recommended.

Decompression laparotomy is considered the definitive treatment for ACS. The threshold for decompression is not well established. Some surgeons use an IAP of 25 mm Hg, others use between 15 and 25 mm Hg, and still others use an APP under 50 mm Hg. After decompression, the abdomen is left open with temporary abdominal wall closure via dressings that bridge the fascia. The disposition for ACS patients is to a surgical ICU.

## KEY POINTS

- Common ED cases to suspect ACS: bowel obstruction, ascites, pancreatitis, trauma, recent major abdominal surgery, massive resuscitation/transfusion, shock, and severe burns.
- Key findings that may indicate ACS: abdominal distention, progressive oliguria, hypercarbia, refractory hypoxemia, and lactic acidosis.
- Use transduced bladder pressures to measure IAP:  $\geq 12$  mm Hg is IAH;  $>20$  mm Hg with new organ dysfunction is ACS.
- In the ED, utilize temporizing medical therapies as indicated.
- Obtain early surgical consultation for possible decompression laparotomy.

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## CARDIOGENIC SHOCK

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KEVIN K. CHUNG, DO

Cardiogenic shock is defined as inadequate tissue perfusion secondary to insufficient cardiac output. It can lead to multiorgan failure, including but not limited to altered mental status, oliguria, renal failure, lactic acidosis, and tissue hypoperfusion. Most commonly, cardiogenic shock occurs in 3% to 8 % of cases with ST elevated myocardial infarctions and 2.5% of non-ST elevation (NSTEMI) cases. Mortality reaches 80% in those with cardiogenic shock following an acute MI. Other than myocardial infarction, any cause of acute left ventricular or right ventricular dysfunction may lead to cardiogenic shock. For example, cardiomyopathies, myopericarditis, stress-induced (tako subo) cardiomyopathy, and acute valvular dysfunction can lead to cardiogenic shock.

The definition of cardiogenic shock has the following criteria: systolic blood pressure < 90 mm Hg or mean arterial pressure (MAP) lower than 30 mm Hg of baseline blood pressure, pulmonary wedge pressure > 15 mm Hg, cardiac index < 2.2 L/min m<sup>2</sup>, adequate or elevated end diastolic filling pressure.

Unfortunately, emergency physicians do not have the luxury of a pulmonary artery catheter to tell us this information, so much of the assessment must come from the physical exam. Decreased tissue perfusion can lead to cool extremities, cyanosis, diminished peripheral pulses, altered mental status, and decreased urine output. Valvular lesions can cause new murmurs, and vascular congestion can lead to an elevated JVP and crackles and rales on auscultation. It is helpful to divide the hemodynamic profiles of patients with heart failure and circulatory dysfunction among four basic profiles that can help you initiate the appropriate therapy.

- 1) **Stable:** no therapy

- 2) **Wet and Warm:** diuretics to reduce congestion
- 3) **Dry and Cold:** low perfusion at rest, although just adequately compensated
- 4) **Wet and Cold:** severe cardiogenic shock requiring vasodilators, inotropic support, diuretics

The pathophysiology of cardiogenic shock can be quite complex, but we will not dive into the intricacies associated with the various etiologies. In essence, cardiogenic shock is classically thought of as ineffective cardiac output leading to hypotension and compensatory tachycardia and increase in systemic vascular resistance due to the release of catecholamines leading to worsening shock physiology. However, the SHOCK (Should We emergently revascularize Occluded coronaries for Cardiogenic Shock) trial revealed that many patients become hypotensive in a mechanism similar to septic shock from the release of cytokines similar to the SIRS response.

The priority of your diagnosis should be determining the presence of a STEMI/NSTEMI as this remains the most common cause with subsequent complications such as papillary rupture and valve dysfunction depending upon the area of infarct. A chest x-ray and echocardiogram can further aid the etiology.

Treatment is focused on stabilizing hemodynamics while treating the underlying cause if it can be reversed. With that said, the priority of therapy should balance the use of inotropes, pressors, diuretics, and obtaining early revascularization in the setting of an MI. The benefits of early revascularization were proven in the SHOCK trial. This includes early thrombolytics, cardiac angiography, and percutaneous coronary intervention (PCI) versus coronary artery bypass graft (CABG).

Medical therapies in the emergency department (ED) can be thought of in the following categories:

### 1) **Acute coronary syndrome drugs**

Antithrombotic therapy with aspirin, clopidogrel, and heparin should be given early in the diagnosis of an acute MI. Nitrates and vasodilators such as nitroprusside should be avoided for refractory hypotension.

### 2) **Vasopressors**

The American College of Cardiology recommends norepinephrine for hypotension. Dopamine has been associated with higher mortality in cardiogenic shock due to being arrhythmogenicity.

### 3) **Inotropic agents**

These medications can be used in conjunction with vasopressors.

Inotropes have a central role in treatment due to the contractile failure that causes cardiogenic shock. Dobutamine has both chronotropy and inotropy and reduces afterload. Milrinone (a phosphodiesterase inhibitor) has less chronotropy and has a greater role in reducing afterload than does dobutamine. Therefore, caution should be used in the ED if the patient does not have an adequate blood pressure, although many argue that it has a greater role than dobutamine in right heart failure. Adjunct therapies may be indicated after discussion with cardiology. These include intraaortic balloon pumps (IABP), ventricular assist devices (Impella), and transvenous pacers.

## KEY POINTS

- Cardiogenic shock can present with hypertension or hypotension.
- Myocardial infarction (MI) is the most common cause of cardiogenic shock, and early revascularization should be a top priority. Especially in the ED, cardiogenic shock can be diagnosed from physical exam and clinical findings. Use the classifications of wet and cold, wet and warm, and dry and cold to guide you in management.
- Although ACS is a common cause, avoid nitroglycerin to avoid refractory hypotension.
- Initiate norepinephrine and dobutamine for vasopressor and inotropic support.
- Remember to involve your cardiology team early as possible in the event that adjunct therapy may need to be initiated.

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# **KNOW WHEN TO ADMINISTER SODIUM BICARBONATE IN THE CRITICALLY ILL POISONED PATIENT**

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**HARRY E. HEVERLING, DO AND TIFFANY C. FONG, MD**

Sodium bicarbonate is an important antidote in the treatment of the critically ill poisoned patient. This contrasts with the general approach to acidosis due to other conditions, where sodium bicarbonate is rarely used and targeting the underlying illness (e.g., sepsis, diabetic ketoacidosis, acute renal failure) is the preferred strategy. Sodium bicarbonate has a vital and specific role in a variety of poisonings, most notably those involving tricyclic antidepressants (TCAs) and salicylates. Among the most important applications of sodium bicarbonate are (1) reversal of sodium channel blockade, (2) alteration of drug distribution and enhancement of elimination, and (3) reversal of life-threatening acidemia.

## **REVERSAL OF SODIUM CHANNEL BLOCKADE**

Blockade of the fast sodium channels may cause fatal cardiotoxic effects, manifested by conduction delays (QRS prolongation, right bundle branch block, wide complex tachycardias) and hypotension. This toxidrome is well noted in poisonings by TCAs, type IA and IC antidysrhythmics (e.g., quinidine, procainamide, flecainide), diphenhydramine, propoxyphene, quinine, carbamazepine, and cocaine.

Sodium bicarbonate is an antidote that reverses the toxicity of sodium

channel blockers by two mechanisms. It creates an alkaline environment that decreases the fraction of the ionized form of the drug available to bind and block the sodium channel, and it increases the number of sodium ions available to move through sodium channels.

The indications for sodium bicarbonate in sodium channel blocker toxicity are QRS duration > 0.10 seconds, ventricular dysrhythmias, or hypotension. Sodium bicarbonate does not effectively treat altered mental status or seizures associated with TCA poisoning; however, it may have utility in limiting further acidemia caused by seizures, which can exacerbate conduction disturbances and dysrhythmias.

## **ALTERATION OF DRUG DISTRIBUTION AND ENHANCEMENT OF ELIMINATION**

Salicylate toxicity has a complex pathophysiology affecting multiple organ systems, and is discussed in greater depth in a separate chapter. Though there is no specific antidote for salicylate poisoning, the administration of sodium bicarbonate is a cornerstone of treatment. Sodium bicarbonate acts both to decrease concentrations of salicylate in target tissues and to enhance elimination of salicylate in the urine via alkalinization.

Salicylate is a weak acid that converts to an ionized form as pH increases. Administration of sodium bicarbonate and subsequent ionization of the drug allows trapping of salicylate in the plasma compartment, as ionized molecules less readily cross cell membranes. Importantly, this decreases binding of drug in the central nervous system (CNS), which causes the greatest morbidity and mortality related to salicylate poisoning. Furthermore, ionization of salicylate in an alkalinized urine environment augments the accumulation and subsequent elimination of salicylate in the urine. Similar principles apply to poisoning by other weak acids, including phenobarbital, chlorpropamide, and chlorophenoxy herbicides.

Indications for sodium bicarbonate in salicylate poisoning include symptoms or signs of systemic toxicity (i.e., CNS or pulmonary manifestations) or significant metabolic acidosis. Some toxicologists also advocate for sodium bicarbonate administration in asymptomatic patients with a salicylate concentration >30 mg/dL. Controversy exists regarding the indications for alkalinization, as excessive alkalemia, hypernatremia, fluid overload, hypokalemia, and hypocalcemia may result. Management with endotracheal intubation and hyperventilation may be employed when necessary (using care to avoid worsening acidemia from respiratory acidosis), and hemodialysis may be required.

# REVERSAL OF SEVERE METABOLIC ACIDOSIS

Toxic alcohol poisoning (e.g., ethylene glycol and methanol) can cause life-threatening acidemia, associated with hemodynamic instability and end organ dysfunction. Though definitive care must include administration of fomepizole or ethanol, and possibly hemodialysis, sodium bicarbonate may be an important temporizing measure to reverse severe acidemia. It is recommended that sodium bicarbonate is administered when arterial pH is  $<7.30$  in the setting of toxic alcohol poisoning. Additional benefits of sodium bicarbonate are the redistribution of toxic metabolites out of target tissues, and enhancement of urinary elimination.

## DOSING OF SODIUM BICARBONATE

Dosing and administration of sodium bicarbonate is similar across multiple poisonings. A hypertonic sodium bicarbonate solution may be initiated as a 1- to 2-mEq/kg intravenous (IV) bolus over 1 to 2 minutes, followed by a continuous infusion. The infusion is created through mixture of three 50 mL ampules of sodium bicarbonate (total of 150 mEq) into a 1-L bag of D5W, and run at approximately twice the maintenance rate. D5W is used because of its hypotonic properties. When combined with sodium bicarbonate, it creates an isotonic solution.

Specifically for TCA poisoning, the initial IV bolus of sodium bicarbonate may be repeated every 5 minutes as needed to achieve narrowing of the QRS interval, and a blood pH of 7.50 to 7.55. Serial electrocardiograms (ECGs), arterial blood gases (ABGs) for pH monitoring, and reassessment of volume status are required as part of ongoing management. The infusion is usually discontinued when improvement in cardiotoxicity, hemodynamic indices, and mental status is achieved.

For a goal of enhanced urinary elimination, as in salicylate poisoning, the infusion should be titrated to a goal urinary pH 7.5 to 8.0 and urine output of 3 to 5 mL/kg/h. ABGs should be followed to ensure serum pH does not exceed 7.55. To ensure successful alkalinization of urine, IV potassium supplementation (through addition of 20 to 40 mEq/L of fluid) may be necessary.

### KEY POINTS

- Though less applicable for the general critically ill patient with

metabolic acidosis, sodium bicarbonate has a vital and specific role as an antidote in the poisoned patient.

- Sodium bicarbonate is a specific reversal agent in cases of poisoning from sodium channel blockers (i.e., TCA and type I antidysrhythmics).
- In salicylate toxicity, sodium bicarbonate favorably redistributes the drug out of vulnerable target tissues, and enhances urinary elimination.
- Sodium bicarbonate can temporize the severe and harmful metabolic acidosis seen in poisoning by toxic alcohols and is used concurrently with definitive care measures.
- Sodium bicarbonate is administered as a 1- to 2-mEq/kg IV bolus, followed by continuous infusion using a mixture of 150 mEq of sodium bicarbonate added to 1 L of D5W. The IV bolus may be repeated as needed in the case of TCA toxicity.

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# THINK BIG VESSELS: VASCULAR CATASTROPHES

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**KEVIN K. CHUNG, DO**

Many would argue that the practice of Emergency Medicine largely revolves around the idea of diagnosing vascular emergencies. There are those diagnoses that require immediate intervention to stabilize hemodynamics in order to preserve tissue perfusion. This chapter will focus on the vascular catastrophes associated with the highest mortality and morbidity that require prompt repair to offer the patient any chance of survival. These include but are not limited to abdominal aortic aneurysm (AAA) rupture and aortic dissection.

## **AAA RUPTURE**

The classic presentation of a ruptured AAA involves an older patient (>60 years old) presenting with sudden onset of abdominal pain, hypotension, and a pulsatile abdominal mass. Roughly 50% of these patients describe an abrupt ripping or tearing pain. AAA should always be on the differential for a patient presenting with syncope. This may seem obvious, but it is important to note that AAAs can have more subtle variations. When one considers AAA in the differential, there are four clinical scenarios that should be considered: acute rupture, aortoenteric fistula, a contained aneurysm, and an incidental aneurysm. Each represent different degrees of severity and have different approaches to management. Missed AAA can be mistaken for renal colic so be wary and perform a thorough physical exam. Retroperitoneal hemorrhage can have flank ecchymosis and periumbilical ecchymosis, and a physician should observe for compression of the femoral nerve causing neuropathy.

Aortoenteric fistulas can arise from previously repaired AAA. One should always consider this in a patient presenting with a GI bleed and hematemesis. The duodenum remains the most common site of fistulization. Ruptured AAA can also be contained into the retroperitoneum. This can be easily missed if an emergency physician does not have this on the differential.

With all of these presentations, a stable patient should have a CT scan. If tolerated, a bedside ultrasound evaluation can be utilized. Prompt vascular access and crossmatched red blood cells should be obtained. Surgical consultation is required for any variation of AAA, and the difference remains in the urgency that it is required. Remember that the primary role of the emergency physician is in identifying an AAA.

## **AORTIC DISSECTION**

Patients with aortic dissection will typically present with chest pain that radiates to the back. They will often be of older age and hypertensive, and the pain is often described as a ripping or tearing pain. Studies have shown that ~70% of patients with ascending involvement have anterior chest pain while 63% have back pain with descending dissections.

The physical exam findings of an aortic dissection will depend on the location of the intimal tear. For example, a dissection that occurs at the aortic arch may involve the subclavian arteries leading to different pulsatile strength and blood pressures between the right and left arm. If it includes the ascending portion of the aorta, a new murmur representing aortic valve insufficiency may occur. It is often easy to forget to consider aortic dissection in a patient with new-onset neurologic deficits. Carotid involvement will present with stroke symptoms, and occlusion of the vertebral blood supply may cause paraplegia, vision loss, or even Horner syndrome.

Diagnosis includes physical exam findings (some of which are listed above), chest x-ray, CT scan if stable, and an EKG that can demonstrate dissection of the coronary artery—typically inferior STEMI from RCA disruption.

Emergent vascular or thoracic surgical consultation is required for ascending aortic dissections, while descending dissections can be evaluated per case, as medical management can often be an option. Aortic dissections should be classified using the Stanford classification or DeBakey to better communicate with the surgeons. Stanford Type A is ascending aorta, and Type B is descending aorta. DeBakey—Type I involves both ascending and

descending aorta, Type II is only ascending aorta, and Type III is descending aorta.

Management of hypertension requires a beta-blocker and vasodilators. It is important to note that a beta-blocker should be administered first to prevent shear forces from reflex tachycardia when starting vasodilators. Most often esmolol (which has a fast half-life) and can be easily titratable, and nitroprusside are used. Calcium channels blockers can also be used. The goal systolic blood pressure should be between 100 and 140 mm Hg.

## KEY POINTS

- The presentation of a patient with AAA can have variations. Do not forget to consider aortoenteric fistula and a contained rupture that will be more subtle and more difficult to diagnose.
- Classify the aortic dissection using Stanford versus DeBakey to communicate with the surgery team.
- Aortic dissections can present with neurologic deficits and should be on the differential when presented with CVA findings and Horner syndrome.
- Obtain surgical consultation early for vascular catastrophes.
- For aortic dissections, start beta-blockers before vasodilators.

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## **STOP THE BLEEDING! NOVEL THERAPIES: REBOA**

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**CASEY LEE WILSON, MD, RDMS**

In penetrating trauma with witnessed loss of vital signs, ED thoracotomy with aorta cross-clamping remains the most common technique for hemorrhagic control. Survival rates for this invasive procedure remain dismal and are currently only around 15%. ED thoracotomy also incurs significant potential risks to the providers who perform them. Vascular hemorrhagic control is obtained by aortic occlusion to reduce bleeding and enhance central aortic pressure, which in turn augments myocardial and cerebral perfusion. Resuscitative endovascular balloon occlusion of the aorta (REBOA) has been designed as an adjunct for controlling hemorrhage in order to sustain vital perfusion until definitive hemostasis can be achieved.

Currently being studied in several civilian trauma centers, REBOA can be placed in about 6 minutes when deployed in a protocolled manner. Placement within the aorta occurs via the common femoral artery, and the depth of placement is guided by the level of trauma as approximated externally. Zone 1 is within the thoracic aorta, relevant for hemorrhagic abdominal trauma, and the balloon catheter is measured externally against the xiphoid process. Zone 3 pertains to the infrarenal aorta for exsanguinating pelvic hemorrhage, measured externally at the level of the umbilicus.

REBOA is a potential emerging alternative to resuscitative thoracotomy (RT) or laparotomy for the rapid control of exsanguinating noncompressible torso trauma. Bridging hemostasis may even obviate the stability and resources currently required to transport a patient to interventional radiology in the instance of pelvic fracture-induced hemorrhage. It is ideal in military and resource poor settings where damage control resuscitation is the key, but



may play a role in the prehospital setting where mortality remains high as a result of hemorrhagic shock.

Noncompressible torso hemorrhage carries almost a 50% mortality rate, and when not treated promptly, patients rapidly progress to cardiovascular collapse and death. Functional outcomes still remain poor for patients who present to the ED in extremis, or even worse, in full arrest. In patients with end-stage hypovolemic shock, REBOA can serve as a salvage method of circulatory support until definitive hemostasis is obtained. REBOA has a considerable hemodynamic effect above the level of aortic occlusion, leading to elevated MAP and cardiac output. Inflation of the balloon allows for improved afterload support and is one argument for REBOA over thoracotomy, as the ability to adjust balloon inflation to generate a particular MAP is desirable. Animal models comparing REBOA to RT demonstrated a higher pH, lower lactate, less fluid and inotropic support, and increased survival in the REBOA group. The length of time for safe continuous aortic occlusion remains to be determined.

Previous studies in the vascular and cardiothoracic surgery literature have demonstrated poor outcomes with aortic-cross-clamping. Spinal cord injury and paralysis are the most dreaded and devastating complications, with a prevalence of up to 23% in the current literature. Preliminary REBOA studies in a swine model suggest that higher and more proximal REBOA placement trends toward a higher degree of spinal cord injury and less functional recovery. This is difficult to extrapolate to potential human outcomes, as there are differences in porcine and human vasculature. The beneficial hemodynamic effect of REBOA must be balanced by the potential untoward metabolic sequelae as a result of ischemia-reperfusion injury. The length of aortic occlusion does correlate with elevated lactate levels and a subsequent inflammatory response, the degree to which this affects cardiopulmonary function is still unknown. Critical care facilities and anticipated organ support are essential in any environment where REBOA is deployed.

Many questions remain unanswered at this time, and future research will likely address indications and contraindications of the procedure, whether REBOA will prove to be useful in thoracic trauma, whether it can compare to RT in unstable patients with abdominopelvic trauma, and whether it can be safely and optimally placed in the prehospital setting. As REBOA techniques and technology advances, practitioners will need to carefully consider the available evidence and resources to determine its applicability in their respective institutions. Early clinical series and large animal studies have shown promise for REBOA as a bridge to definitive hemostasis. No doubt, this is an exciting area of investigation for the proactive management of

noncompressible torso hemorrhage.

## KEY POINTS

- To date, RT with open aortic cross-clamping and internal cardiac massage remains the current treatment for noncompressible hemorrhage in the setting of traumatic cardiopulmonary arrest.
- Early REBOA data and ongoing studies suggest that procedure-related deaths and major complications are minimal, and it is an emerging concept that may hold promise as an alternative treatment for noncompressible torso hemorrhage.
- This technique is still in its infancy, and its clinical niche might be best served in the military or prehospital setting, as these tend to be low resource settings with high mortality rates as a result of hemorrhage that occurs prior to hospital arrival.
- REBOA may be a “future consideration” for emergency physicians to consider as an alternative to ED thoracotomy for a patient with exsanguinating hemorrhage below the diaphragm leading to extremis or cardiac arrest.
- No high-level data currently exists to support the widespread promotion of REBOA use at this time. Further research as well as institution-dependent resources and expertise will need to be considered prior to widespread application and use of REBOA.

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# **AVOID THE TUBE! NONINVASIVE VENTILATION STRATEGIES**

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**RODICA RETEZAR, MD, MPH**

## **INTRODUCTION**

The emergency physician (EP) is always on the front line when dealing with patients with acute respiratory failure. Prompt intervention to optimize the oxygenation and ventilation processes of patients in distress is critical in order to avoid serious complications and death. Over the last several decades, noninvasive ventilation (NIV) has been shown to be a valuable asset when managing patients in respiratory failure due to a variety of conditions.

## **MODES OF NONINVASIVE VENTILATION**

The two forms of NIV often used in the emergency department (ED) are continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP). CPAP delivers a constant amount of pressure throughout both the inspiratory and expiratory phases of the respiratory cycle. This improves oxygenation by inflating collapsed alveoli (alveolar recruitment), redistributing pulmonary fluid, and diminishing ventilation-perfusion mismatch. Furthermore, CPAP increases intrathoracic pressure resulting in a decreased preload which in many cases can lead to improved left ventricular function. BiPAP cycles between delivering a higher airway pressure during inhalation (inspiratory positive airway pressure [iPAP]) and a lower airway pressure during exhalation (expiratory positive airway pressure [ePAP]). The ePAP component of BiPAP is equivalent in mechanism to CPAP, augmenting oxygenation in patients with hypoxic respiratory failure. A higher iPAP compared to ePAP causes a differential gradient between the

iPAP and ePAP thereby facilitating airflow and ventilation and increasing carbon dioxide elimination in patients with hypercarbic respiratory failure.

## PATIENT SELECTION

Contraindications for NIV include cardiac arrest, hemodynamic instability, inability to protect the airway, need for immediate intubation, upper airway obstruction, severe gastrointestinal bleeding, vomiting, and facial trauma. The ideal candidate for NIV is a patient who is alert, able to follow commands, handles his/her secretions, and is able to tolerate the ventilator. NIV should be initiated as soon as possible in patients who exhibit significant work of breathing to prevent worsening hypercarbia and hypoxia as a result of respiratory insufficiency.

Once the decision has been made to place patient on NIV, the appropriate mask and size must be chosen. The oronasal mask, fitted to include adequate coverage for the mouth and the nose, is the type most commonly used in the ED. The oronasal mask has been shown to be more effective than the nasal mask in patients with acute respiratory failure. After the patient is fitted with the correct-sized mask, the NIV mode and the pressure level is selected. It is best to start with lower pressure(s) to allow the patient to adapt to the mask and ventilator. The pressure(s) can gradually be increased to optimize the oxygenation and ventilation processes.

Oxygenation can be improved by increasing the amount of oxygen supplied (fraction of inspired oxygen or  $\text{FiO}_2$ ). Alternatively, this can also be achieved by increasing the pressure in CPAP (or the ePAP in BiPAP mode) to levels of 10 to 15 cm  $\text{H}_2\text{O}$ . This serves to increase positive end expiratory pressure and in turn functional reserve capacity (PEEP and FRC). Increases in iPAP to levels of 20 to 25 cm of  $\text{H}_2\text{O}$  while keeping ePAP constant for patients on BiPAP can improve ventilation and hypercarbia. After initiation of NIV, patients should be closely monitored to determine appropriate response to the intervention. The key components of reevaluation include oxygen saturation, heart rate, blood pressure, respiratory rate, mental status, cardiopulmonary exam, and blood gas values.

If the patient is not tolerating NIV, it may be necessary to change to a different mask, mode, or pressure setting before calling it a treatment failure and proceeding with endotracheal intubation. Additionally, pharmacologic agents such as low dose, short-acting opioids or benzodiazepines can be used to alleviate the anxiety and discomfort related to NIV.

# INDICATIONS

## ***Congestive Heart Failure and Chronic Obstructive Pulmonary Disease***

There is robust evidence to support that NIV decreases the need for intubation, shortens hospital and ICU stays, and improves mortality in patients with congestive heart failure (CHF) and chronic obstructive pulmonary disease (COPD) exacerbations. Some of the earlier literature pointed toward an increased myocardial infarction rate in patients with CHF placed on BiPAP compared to those on CPAP. However, this finding has not been demonstrated in subsequent meta-analysis and Cochrane reviews.

## ***Asthma***

While the data on use of NIV in patients with asthma exacerbation are mixed when it comes to reduction in intubation and mortality, it does show that NIV improves pulmonary function and decreases hospital and ICU length of stay. A short trial of NIV is a reasonable choice in asthmatic patients given the significant risk of barotrauma and hyperinflation associated with intubation.

## ***Pneumonia/Immunocompromised Patients***

The use of NIV in community-acquired pneumonia (CAP) is controversial. A recent Cochrane review suggests that NIV may be more beneficial in CAP, but there are insufficient studies to be conclusive. Earlier studies suggested improved mortality and lower intubation rates in these patients, while some recent reviews suggest a high NIV failure rate and worsening mortality in patients with significant respiratory distress. The current recommendations are that patients with underlying pulmonary or cardiac disease may be more appropriate candidates for early NIV when diagnosed with CAP. There are several reviews that have demonstrated that NIV reduces mortality and intubation rates in patients with pneumonia especially if they have underlying COPD, as well as in immunocompromised patients with infiltrates and fever.

## ***Blunt Chest Trauma***

Patients with blunt chest trauma who receive early NIV may have decreased intubation rate, ICU stay, and mortality. Patients included in these studies were diagnosed with pulmonary contusions, rib fractures, flail chest, or sternal fractures. The data seem to support the use of NIV in patients who are

hemodynamically stable, hypoxic but have not yet developed fulminant respiratory failure.

## ***Preoxygenation Prior to Intubation***

Patients who require intubation should be preoxygenated with 100% oxygen delivered by a nonrebreather in order to prevent desaturation during the apneic period. There are times when this method is not enough to adequately raise the saturation level prior to induction. Recent studies have shown that patients who are preoxygenated with NIV have fewer desaturations during the intubation process when compared to patients receiving 100% oxygen via bag-valve mask.

### **KEY POINTS**

- If the patient is an appropriate candidate, initiate NIV as soon as possible.
- NIV provides the most benefit in patients with COPD and CHF exacerbation.
- A short trial of NIV is appropriate for patients with certain other conditions that compromise oxygenation or ventilation.
- IPAP increased relative to ePAP increases ventilation, thereby decreasing CO<sub>2</sub>. Increasing ePAP is similar to providing PEEP/CPAP and helps to increase oxygenation.
- Patients on NIV need to be frequently reevaluated to determine treatment success. Worsening clinical status or lack of improvement is an indication to escalate to endotracheal intubation.

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## BE WARY OF INTUBATION IN THE ASTHMA PATIENT

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**DANIEL B. SAVAGE, MD, MPH**

According to the Centers for Disease Control, over 24 million Americans carry a diagnosis of asthma, which works out to >7% of the U.S. population! Given this statistic, it should come as no surprise that asthma is the most common chronic medical condition among pediatric patients. As such, emergency department visits due to asthma exacerbations are extremely common.

An asthma exacerbation can be triggered by a wide range of environmental stimuli but ultimately consists of bronchospasm, airway inflammation, and increased airway secretions. This triad leads to outflow obstruction and an inability to fully exhale, otherwise known as air trapping. The mainstays of therapy for asthma exacerbations include systemic corticosteroids, inhaled beta-2 agonists, and inhaled anticholinergics. However, when asthmatics fail to improve with these therapies, they are considered to have acute severe asthma or status asthmaticus. Early recognition and aggressive management of status asthmaticus in the emergency department is key—as this condition can be life threatening.

An asthma exacerbation is a clinical diagnosis based off of history and physical exam. Patients will often have an exaggerated inspiratory to expiratory ratio, diffuse end-expiratory wheezing, and increased work of breathing. Remember the degree of wheezing does not always correlate with the severity of the exacerbation. Be wary of the quiet lungs—which often means that the patient is only moving a minimal amount of air.

Asthmatics who present to the ED with an acute exacerbation of their underlying obstructive lung disease are all given inhaled beta-2 agonists



(albuterol) and inhaled anticholinergics (ipratropium). Additionally, early administration of systemic corticosteroids is important, as this medical intervention does not have any appreciable clinical effect for ~6 hours. If inhaled medications are not readily available or not tolerated, subcutaneous systemic beta-agonist therapy (terbutaline 0.45 mg q20min × 3 doses) as well as subcutaneous epinephrine (1:1,000 0.3 to 0.5 mg q20min × 3 doses) can be given. Systemic bronchodilation in the form of intravenous magnesium sulfate (2 g over 20 minutes) can serve as an adjunct to inhaled medications in status asthmaticus.

Asthmatics in status who have received the previously mentioned medical therapies and are on continuous nebulized albuterol warrant close monitoring. If at all, possible intubation should be avoided as it has been shown to increase mortality and lead to worsening barotrauma, breath stacking, and hyperinflation. However, worsening hypoxia/hypercarbia, declining mental status, muscle fatigue, and apnea are all indications to pursue more aggressive treatment modalities in the form of noninvasive positive pressure ventilation (NIPPV) or mechanical ventilation.

NIPPV in status asthmaticus is a controversial topic in emergency medicine that has not been well studied. Despite its continued clinical use, a 2012 Cochrane Review was inconclusive in regard to its overall benefits. Theoretically, NIPPV can decrease overall work of breathing and fatigue as well as improve gas exchange. However, NIPPV should not be used in patients who are agitated, who are vomiting, or who have profuse secretions or depressed mental status. At a minimum, NIPPV can serve as a bridge to intubation by providing ventilatory support and preoxygenation as the physician prepares to establish a definitive airway.

Once the clinical decision is made to intubate a patient in status asthmaticus, ketamine can be considered as the induction agent (as ketamine can provide further bronchodilation). Some authors suggest pretreatment with an anticholinergic agent such as ipatropium to help reduce secretions associated with ketamine. Post intubation, asthmatics are prone to hypotension due to increased intrathoracic pressures from air trapping and the positive pressure of mechanical ventilation, as such make sure to have IV fluids hanging. In order to limit hyperinflation, it is important to reduce the patient's minute ventilation (respiratory rate + tidal volume). Start at a tidal volume of 6 cc/kg (lower if plateau pressures are not <30 cm H<sub>2</sub>O). The inspiratory to expiratory ratio should be dramatic (on the order of 1:5), so that patients are able to fully exhale prior to receiving another breath. It is tempting in hypercarbic patients to titrate up the respiratory rate to blow off CO<sub>2</sub>; however, this will only serve to worsen hyperinflation and can lead to

circulatory collapse!

## KEY POINTS

- If nebulized medications are not readily available or are not well tolerated—remember subQ terbutaline and epinephrine.
- Avoid intubation if at all possible as it has been shown to increase mortality and worsen barotrauma/hyperinflation.
- Indications to intubate: worsening hypercarbia/hypoxia, declining mental status, muscle fatigue, apnea.
- Consider ketamine as your induction agent for RSI in an asthmatic.
- Hypercarbia should be tolerated and treated with a low RR in intubated patients—so that asthmatics are able to fully exhale before triggering another breath.

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# **SECTION II**

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# **CRITICAL CARE**

# STOP USING BENZODIAZEPINES TO SEDATE YOUR CRITICALLY ILL INTUBATED PATIENT

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**KRYSTLE SHAFER, MD AND LILLIAN L. EMLET, MD,  
MS**

Care of the intubated emergency department (ED) patient does not end with securing the endotracheal tube (ETT) and the initial mechanical ventilator settings. It is critical to provide patients with analgesia and sedation. Intubated patients experience pain and anxiety from a variety of sources, including the presence of an ETT, lung-protective ventilator settings, placement of catheters, and even routine nursing care. Pain and agitation elevate levels of catecholamines, which induce vasoconstriction, impair myocardial perfusion, and ultimately reduce cardiac output.

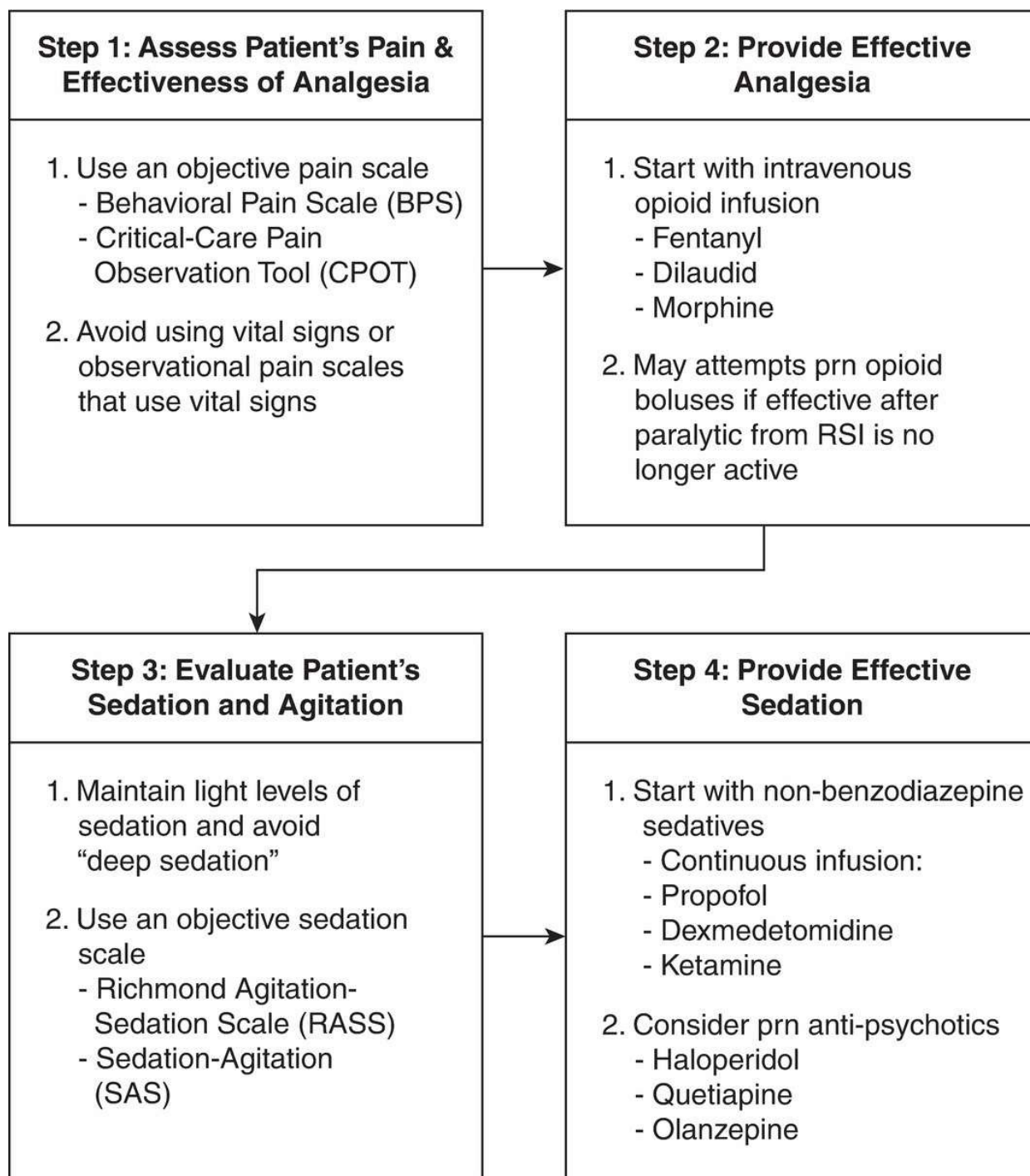
Recent literature highlights that many intubated ED patients receive inadequate, or no, analgesic or sedative medications. A 2014 retrospective review reported that 18% of newly intubated ED patients did not receive any postintubation sedatives despite receiving a long-acting neuromuscular blocking agent. Even if an analgesic or sedative is administered in the ED, it is often delayed.

Benzodiazepines are frequently used for sedation in critically ill intubated patients. Importantly, benzodiazepines are associated with an increased incidence of delirium, and increased intensive care unit (ICU) length of stay, and increased mortality. The elderly patient is even more sensitive to the adverse effects of benzodiazepines. As such, a recent clinical guideline on the management of pain and agitation in critically ill ICU patients recommends avoiding benzodiazepines for the treatment of

agitation.

For the critically ill intubated patient, begin with an analgesic medication rather than a benzodiazepine. An “analgo-sedation” strategy, whereby the analgesic is given first followed by a sedative, has been shown to decrease the duration of mechanical ventilation and decrease ICU length of stay. Opioid medications are considered first-line agents for postintubation analgesia. Although no opioid has been shown to be superior, the authors prefer fentanyl. Fentanyl is a highly potent opioid that has a quick onset of action and can also be administered as a continuous infusion. In contrast to morphine, fentanyl does not cause histamine release, making it less likely to cause hypotension.

If agitation persists despite adequate analgesic medication, evaluate the patient for hypoglycemia, hypotension, worsening hypoxia, and withdrawal of a chronic medication. It is also important to examine the ventilator to ensure that settings are correct. Though delirium is less likely in the ED, it should be considered. Use of an antipsychotic medication (i.e., haloperidol, quetiapine, olanzapine) may be effective. Pain and delirium should be treated first before proceeding to sedative medications ([Figure 37.1](#)).



Protocols to assess agitation in the critically ill patient have been shown to reduce the duration of mechanical ventilation, decrease infection rates, and reduce 30-day mortality. The most commonly used protocol is the Richmond Agitation Sedation Scale (RASS). These protocols should be used to titrate sedative medications in the intubated patient. Current guidelines recommend propofol or dexmedetomidine for sedation. Propofol has a rapid onset of action and can be especially useful in patients in status epilepticus. Propofol also has quick offset, allowing the provider to easily perform a neurologic evaluation. The most common side effect of propofol is hypotension, which can usually be treated with intravenous fluids. At high doses this medication

can cause propofol infusion syndrome and hypertriglyceridemia. Propofol should be avoided in patients with pancreatitis and egg or soybean allergies.

Dexmedetomidine is an  $\alpha$ -2 agonist that has an anxiolytic effect, but with minimal respiratory depression. Dexmedetomidine has been shown to be noninferior when compared to midazolam and propofol for sedation. When compared to benzodiazepines, patients who receive dexmedetomidine have decreased delirium and decreased duration of mechanical ventilation. Dexmedetomidine can be continued as an infusion even after extubation. The most common side effects include hypotension and bradycardia.

Ketamine is a noncompetitive NMDA antagonist that provides both analgesia and anxiolysis and can be used for sedation. It can be administered as a bolus or continuous infusion. Unlike dexmedetomidine or propofol, ketamine is less likely to cause bradycardia. In contrast, the sympathomimetic effects of ketamine often increase blood pressure and heart rate. Traditionally, it has been taught that ketamine results in increased intracranial pressure. However, current literature suggests that any increase in intracranial pressure is offset by an improvement in cerebral perfusion pressure. Initiating a ketamine infusion for sedation after endotracheal intubation should be done in discussion with the ICU team who will continue to care for the patient. Propofol, dexmedetomidine, and ketamine are all excellent choices to treat agitation in the critically ill. Benzodiazepines should be avoided, or used as a last resort, for sedation in the intubated ED patient.

## KEY POINTS

- Treat pain and agitation in the intubated ED patient.
- Use validated scales, such as RASS, to objectively assess pain and agitation.
- Treat pain first.
- Avoid benzodiazepines—they cause delirium and increase mortality and ICU length of stay.
- Consider propofol or dexmedetomidine as sedatives in critically ill patients.

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# MONITOR THE PLATEAU PRESSURE IN INTUBATED ED PATIENTS

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**BRIAN J. WRIGHT, MD, MPH**

Critically ill patients are frequently intubated and initiated on invasive mechanical ventilation (IMV). In fact, ~3% of hospital admissions require IMV. Emergency department (ED) patients are placed on IMV for many different reasons. Regardless of the reason, providing safe mechanical ventilation is a core tenet of resuscitative medicine. Recent literature suggests that there is opportunity for improvement in ED IMV management. This has led to an emphasis on providing lung protective ventilation strategies (LPVS) in the ED. Critical components of LPVS include correctly measuring and interpreting peak inspiratory pressure (PIP) and plateau pressure (Pplat).

Improper use of IMV can cause harm. The concept of ventilator-induced lung injury (VILI) was first described in the 1970s. The proposed mechanisms of VILI include barotrauma, volutrauma, atelectrauma, and biotrauma. Barotrauma occurs when the lung is exposed to excessively high inspiratory pressures, resulting in alveolar rupture. Volutrauma is caused by excessive tidal volumes (Tv) and results in stretching of the alveoli. Atelectrauma is caused by the repeated opening and closing of alveoli during inspiratory and expiratory cycles. This continuous shear and strain injury activates inflammatory mediators, resulting in pulmonary and extrapulmonary organ dysfunction, known as biotrauma.

Numerous studies have demonstrated the benefit of LPVS in the majority of patients requiring IMV. In a landmark trial, an IMV strategy that targeted a Tv of 4 to 6 mL/kg of ideal body weight (IBW), a Pplat <30 cm H<sub>2</sub>O, and the judicious use of positive end-expiratory pressure and FiO<sub>2</sub> ([Table 38.1](#))

demonstrated a decrease in mortality when compared to IMV strategy that targeted a  $T_v$  of 10 to 12 mL/kg of IBW in patients with acute respiratory distress syndrome (ARDS). LPVS in the ARDSNet trial was associated with absolute risk reduction of 7% and a number needed to treat of 14. The adoption of LPVS has unfortunately been slow in the ED and intensive care unit. Current evidence also suggests that in patients without ARDS,  $T_v$  should be set to 6 to 8 mL/kg IBW.

**TABLE 38.1 PEEP AND  $FiO_2$**

**LOWER PEEP/HIGHER  $FiO_2$**

|         |     |     |     |     |     |       |     |     |
|---------|-----|-----|-----|-----|-----|-------|-----|-----|
| $FiO_2$ | 0.3 | 0.4 | 0.4 | 0.5 | 0.5 | 0.6   | 0.7 | 0.7 |
| PEEP    | 5   | 5   | 8   | 8   | 10  | 10    | 10  | 12  |
| $FiO_2$ | 0.7 | 0.8 | 0.9 | 0.9 | 0.9 | 1.0   |     |     |
| PEEP    | 14  | 14  |     |     |     | 18–24 |     |     |

**HIGHER PEEP/LOWER  $FiO_2$**

|         |     |         |     |     |     |     |     |     |
|---------|-----|---------|-----|-----|-----|-----|-----|-----|
| $FiO_2$ | 0.3 | 0.3     | 0.3 | 0.3 | 0.3 | 0.4 | 0.4 | 0.5 |
| PEEP    | 5   | 8       | 10  | 12  | 14  | 14  | 16  | 16  |
| $FiO_2$ | 0.5 | 0.5–0.8 | 0.8 | 0.9 | 1.0 | 1.0 |     |     |
| PEEP    | 18  | 20      | 22  | 22  | 22  | 24  |     |     |

It is important for the emergency physician to monitor and measure the PIP and Pplat in any patient receiving IMV. Knowing both values will give the clinician important data to aid in patient management. PIP reflects the sum of large airway resistance and pulmonary compliance. Most modern ventilators readily give the PIP ([Figure 38.1](#)). An elevation in the PIP with a low or normal Pplat suggests a problem with large airway resistance. The differential diagnosis includes



Image courtesy of John Greenwood, MD

**Figure 38.2** This figure shows the Peak Inspiratory Pressure (PIP) or Ppeak.

- Endotracheal tube obstruction or kinking
- Foreign body aspiration
- Bronchospasm
- Airway compression
- Ventilator dyssynchrony
- Excessive gas flow rate

Pplat is a measure of both the alveolar pressure and lung compliance. In a volume assist-control mode of IMV, Pplat is obtained by stopping flow at the end of inspiration for ~1 to 2 seconds. With no flow of air from the ventilator, the pressure across the pulmonary system equilibrates, allowing the ventilator to estimate pressure at the level of the alveoli ([Figure 38.2](#)). Importantly, patients must be in synchrony with the ventilator to perform the inspiratory hold. A common error is to attempt an inspiratory pause while the patient is receiving a pressure-regulated volume control (PRVC) mode of IMV. Measurements done in this mode provide an inaccurate estimation of the true Pplat. When dealing with a patient with an elevation in both PIP and

Pplat, the differential diagnosis includes the following:



**Figure 38.2** This figure shows the PIP and an Inspiratory Pause maneuver to determine Pplat.

- Pneumothorax
- Main stem intubation
- Increasing pulmonary edema
- Mucus plug and atelectasis
- Pleural effusion
- Increased extrathoracic pressure (i.e., chest wall edema, abdominal compartment syndrome, ascites)
- Excessive tidal volumes

In the setting of excessive PIP and Pplat, a chest radiograph or lung ultrasound should be performed to exclude these conditions. After these complications are excluded, Tv should be reduced to target Pplat of 30 cm H<sub>2</sub>O or less. Inducing a mild respiratory acidosis (pH > 7.2) is acceptable in order to achieve safe alveolar distending pressures. This strategy, known as permissive hypercapnia, should be pursued provided that there are no contraindications to lowering pH (i.e., refractory acidosis, severe

hemodynamic instability, elevated intracranial pressure).

When managing a patient on IMV, it is important to remember the principles of LPVS and the concept of VILI. Although a lifesaving intervention, IMV can cause harm if done incorrectly. The emergency medicine physician must be able to measure and interpret the causes of excessive PIP and Pplat in the patient requiring IMV. In doing so, the clinician can rapidly identify an emergent complication and prevent secondary injury.

## KEY POINTS

- Mechanical ventilation can cause harm.
- Measuring PIP and Pplat is critical to providing effective ED IMV.
- Pplat is measured by an inspiratory pause maneuver and reflects the pressure distending the alveoli.
- An elevated PIP with a low Pplat indicates an obstructive process, whereas an elevated PIP and Pplat signifies poor lung compliance and probable progression of an intrinsic lung process.
- Pplat should be kept <30 mm H<sub>2</sub>O to minimize the risk of VILI.

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# **FORGET CVP! USE DYNAMIC MARKERS OF VOLUME RESPONSIVENESS TO GUIDE FLUID RESUSCITATION IN THE CRITICALLY ILL PATIENT**

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**MICHAEL ALLISON, MD**

Fluid resuscitation is the cornerstone therapy for emergency department (ED) patients in shock. The difficulty with fluid resuscitation in the critically ill patient is that up to 50% of patients will not augment their cardiac output (CO) with intravenous fluid (IVF) administration. Deciding whether to continue IVFs based solely on noninvasive blood pressure measurements places patients at risk of overresuscitation. A markedly positive fluid balance in critically ill patients is associated with prolonged mechanical ventilation, acute kidney injury, and increased mortality.

Central venous pressure (CVP) measurements have been the traditional method of monitoring fluid resuscitation in critically ill patients. CVP was a central component of early goal-directed therapy for patients with severe sepsis and septic shock. Recent literature has demonstrated that using CVP to guide IVF therapy has the same predictive ability as a coin toss. In fact, the ProCESS, ARISE, and ProMISe trials found no benefit to CVP-driven sepsis protocols when early IVFs and timely antibiotics were administered. The adoption of dynamic methods of hemodynamic monitoring has changed the landscape of IVF therapy in patients undergoing acute resuscitation. These methods use the variations in CO or stroke volume (SV) to assess the

likelihood that a patient will benefit from IVFs. Dynamic markers of volume responsiveness should become the standard in resuscitation in the ED.

## **ARTERIAL WAVEFORM ANALYSIS**

One method of determining a patient's volume responsiveness is pulse pressure variation (PPV) through arterial waveform analysis. PPV with respiration has been shown to be predictive of volume responsiveness. In addition to PPV, stroke volume variation (SVV) with respiration can be used to guide IVF therapy. The sensitivity and specificity of these techniques are >80%. Commercial devices that measure the area under the arterial pulse waveform can provide an estimation of SV.

## **ULTRASOUND ASSESSMENT OF THE INFERIOR VENA CAVA**

Point-of-care ultrasound (POCUS) can be used to determine whether patients may benefit from additional IVFs. Assessment of respirophasic changes in the diameter of the inferior vena cava (IVC) and SVV, estimated by the aortic blood flow velocity time integral (VTI), are two POCUS measurements that yield information regarding volume responsiveness. POCUS IVC measurements are integrated into ultrasound training programs for emergency and critical care physicians. The IVC undergoes conformational changes as intrathoracic pressure rises and falls. These changes provide information about volume responsiveness in ventilated patients, along with estimates of CVP in nonventilated patients. A 15% collapse, or greater, of the maximal diameter of the IVC suggests that additional IVFs will augment CO. It is important to note that IVC ultrasound measurements are not reliable in patients on low tidal volume ventilation (<8 mL/kg) and in patients with arrhythmias or right ventricular failure. In addition, it has limited utility in patients who are spontaneously breathing (even those spontaneously breathing on mechanical ventilation).

Blood flow through the aortic outflow track is also subject to changes in intrathoracic pressure. Transthoracic echocardiography (TTE) with Doppler measurement of blood flow estimates SV and can be used to determine volume responsiveness. These Doppler measurements are typically obtained in the apical four-chamber view.

## **END-TIDAL CARBON DIOXIDE**

The change in end-tidal carbon dioxide (ETCO<sub>2</sub>) concentration can be used with fluid boluses to determine volume responsiveness. ETCO<sub>2</sub> is determined by pulmonary blood flow, CO<sub>2</sub> production, and minute ventilation. When ventilation and production of CO<sub>2</sub> are constant, pulmonary blood flow can be used as a surrogate for CO. A 3% increase in ETCO<sub>2</sub> after a fluid bolus is a highly specific finding for volume responsiveness. When used with passive leg raise (PLR) (described below), a 5% increase in ETCO<sub>2</sub> has both high sensitivity and specificity for volume responsiveness.

## PASSIVE LEG RAISE

A PLR maneuver provides patients with a reversible volume challenge of 200 to 300 mL of blood from the lower extremities. It is performed by placing patients in a semirecumbent position (head of bed to 45 degrees), rapidly lowering them to a supine position, and then raising the feet to 45 degrees. This produces a fast-acting change in SV and CO that can be measured by one of the above methods, with the exception of SPV and PPV. The benefit of PLR is that it can be used in spontaneously breathing patients and in patients with arrhythmias. The limitation of this technique is the need to rapidly assess SV or CO with a PLR. This can be accomplished with an arterial catheter-based assessment of SV or CO. Alternatively, point-of-care Doppler ultrasonography can also be used to measure changes in SV or CO. Simply evaluating changes in systolic blood pressure with a PLR is not reliable.

### KEY POINTS

- Fifty percent of critically ill patients will not improve their cardiac output in response to a volume challenge.
- CVP does not predict volume responsiveness in the critically ill.
- Arterial waveform analysis can determine volume responsiveness in patients in sinus rhythm and who have no spontaneous respirations.
- IVC ultrasound measurements are not reliable in the setting of low tidal volume ventilation, right ventricular failure, or spontaneous breathing.
- PLR provides a reversible volume challenge to assess volume responsiveness.



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# CONSIDER ABDOMINAL COMPARTMENT SYNDROME IN PATIENTS WITH REFRACTORY HYPOTENSION

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**CINDY H. HSU, MD, PhD**

A 54-year-old male with a history of significant alcohol abuse arrives to your emergency department (ED) with severe abdominal pain and hypotension. You quickly diagnose him with acute pancreatitis and began crystalloid fluid resuscitation. After several liters of intravenous fluid and vasopressor therapy, the patient remains hypotensive with increasing abdominal distention, tachypnea, and oliguria.

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are increasingly recognized in critically ill ED and intensive care unit patients. Unfortunately, the diagnosis of IAH or ACS is often delayed, or missed, due to the complexity of the clinical presentation and limitations of the physical exam. Delays in the diagnosis of IAH or ACS lead to significant increases in morbidity and mortality.

In 2004, a group of international physicians and surgeons created the World Society of the Abdominal Compartment Syndrome (WSACS) in order to promote research, review literature, and formulate guidelines for the detection, prevention, and treatment of IAH and ACS. The critical component in the recognition and diagnosis of IAH and ACS is the detection of an elevated intra-abdominal pressure (IAP). IAP is the steady-state pressure within the abdominal cavity. The normal IAP is ~5 to 7 mm Hg. IAH is defined by a sustained, or repeated, elevation in IAP that is greater

than or equal to 12 mm Hg. IAH is classified into four categories: Grade I, IAP 12 to 15 mm Hg; Grade II, IAP 16 to 20 mm Hg; Grade III, IAP 21 to 25 mm Hg; and Grade IV, IAP >25 mm Hg.

ACS is a sustained IAP >20 mm Hg with new organ dysfunction or failure. ACS may be classified as primary, secondary, or recurrent, depending on the cause and duration. *Primary* ACS is characterized by IAH that results from an abdominopelvic cause (trauma or post–abdominal surgery) and frequently requires surgical or interventional radiologic treatment. *Secondary* ACS refers to conditions that do not originate from the abdominopelvic region and is often seen in patients requiring massive fluid resuscitation. *Recurrent* ACS represents the recurrence of ACS despite resolution of a prior episode that was treated with medical or surgical therapy.

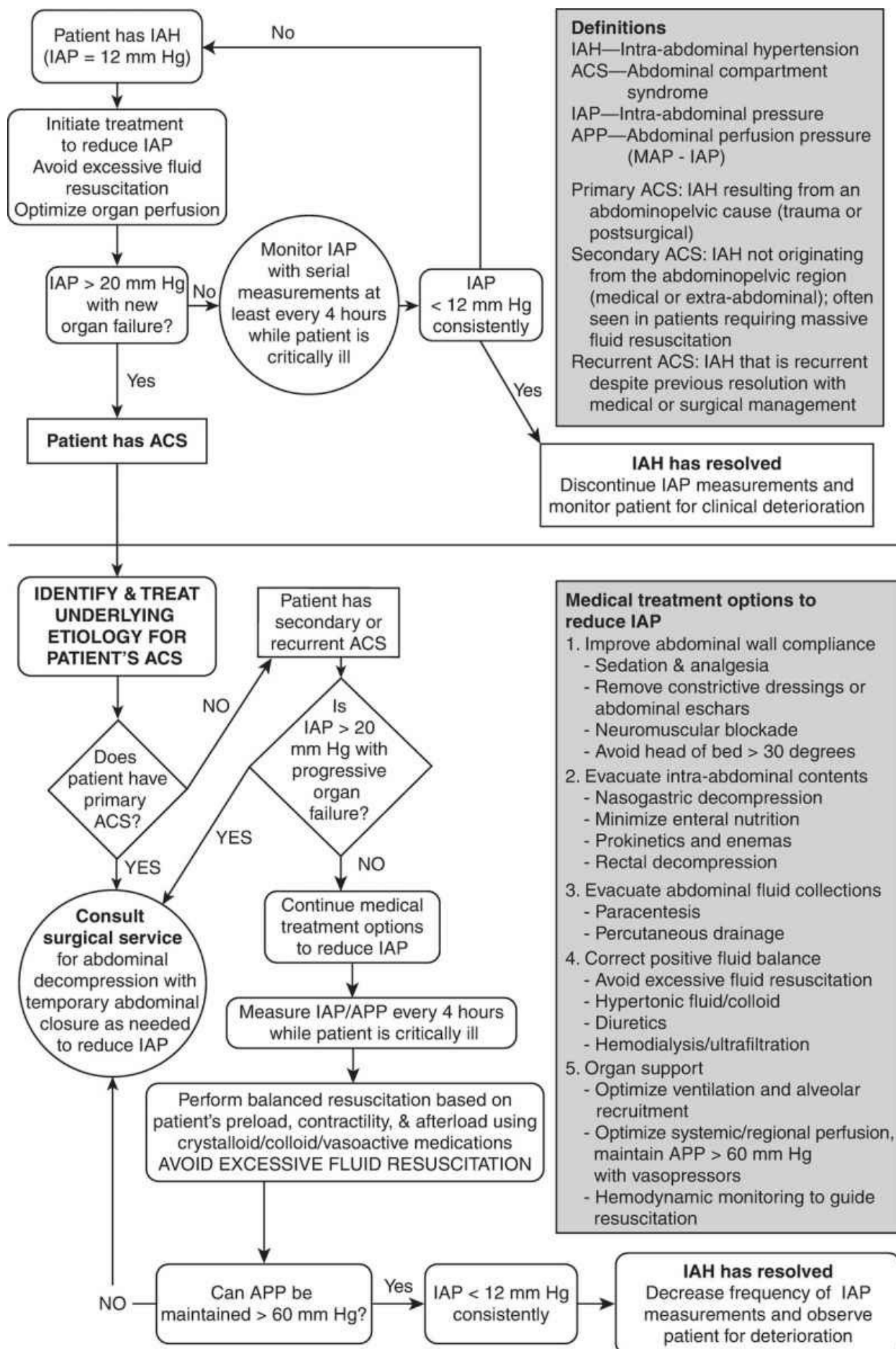
Trans–bladder pressure measurements are simple and economical and remain the gold standard to determine IAP. Bladder pressure measurements should be performed at the end of expiration with the patient in a supine position. The transducer should be zeroed at the iliac crest in the midaxillary line. Approximately 25 mL of saline is then instilled into the bladder. To allow for detrusor muscle relaxation, record IAP measurements 30 to 60 seconds after saline instillation. It is also important to ensure there are no abdominal muscle contractions, as these can falsely elevate the IAP. Measure IAP approximately every 4 hours; however, measurements can be performed at shorter intervals if the IAP is >12 mm Hg. The WSACS recommends a protocolized method for monitoring and measuring IAP if the patient has two or more risk factors for ACS ([Table 40.1](#)) or has new or progressive organ failure.

**TABLE 40.1 RISK FACTORS FOR IAH AND ACS**

1. Diminished abdominal wall compliance
  - (a) Abdominal surgery
  - (b) Major trauma/burns
  - (c) Prone positioning, head of bed >30 degrees
  - (d) Acute respiratory failure with elevated intrathoracic pressure
  - (e) Obesity
2. Increased intraluminal contents
  - (a) Gastroparesis/gastric distention
  - (b) Ileus
  - (c) Colonic pseudo-obstruction
  - (d) Volvulus
3. Increased intra-abdominal contents
  - (a) Hemoperitoneum/pneumoperitoneum
  - (b) Intra-abdominal infection/abscess
  - (c) Intra-abdominal or retroperitoneal tumors
  - (d) Massive incisional hernia repair
  - (e) Liver dysfunction/cirrhosis with ascites
  - (f) Peritoneal dialysis
4. Capillary leak/fluid resuscitation
  - (a) Acidosis (pH < 7.2)
  - (b) Damage control laparotomy
  - (c) Hypothermia (core temperature < 33°C)
  - (d) Massive fluid resuscitation (>5 L/24 h) or transfusion (>10 units blood/24 h)
  - (e) Peritonitis
  - (f) Acute pancreatitis
  - (g) Pneumonia
  - (h) Sepsis/bacteremia
  - (i) Shock or hypotension
  - (j) Coagulopathy

Adapted from Kirkpatrick AW, Roberts DJ, De waele J, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: Updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. *Intensive Care Med.* 2013;39(7):1190–1206.

Once IAH or ACS is diagnosed, appropriate medical or surgical treatments should be implemented to decrease the IAP. Medical treatments recommended by the WSACS to reduce IAP are listed in [Figure 40.1](#). If IAP remains >20 mm Hg with persistent organ dysfunction despite medical therapy, surgical therapy with abdominal decompression should be promptly performed.



**Figure 40.1** IAH/ACS management algorithm. *Adapted from*

Kirkpatrick AW, Roberts DJ, De waele J, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: Updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. *Intensive Care Med.* 2013;39(7):1190–1206.

## KEY POINTS

- Physical exam is insensitive for the diagnosis of IAH or ACS.
- Measure transbladder pressure for high-risk patients to detect IAH and ACS.
- Determine the type of IAH to guide treatment for IAP reduction.
- Prompt medical or surgical interventions are critical for the reduction of IAP and prevention of ACS.
- Refractory ACS warrants prompt surgical abdominal decompression.

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# KNOW THE THRESHOLDS FOR RED BLOOD CELL TRANSFUSION IN THE CRITICALLY ILL

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**MICHAEL C. SCOTT, MD**

While it is not the only state that defines a critically ill patient, shock is considered by many to be the prototypical derangement of the critically ill. With our bedside treatment goals so focused on blood pressure and cardiac output, it can be easy to forget that shock at its most basic level is defined by a deficit between oxygen delivery and oxygen demand at the cellular level. In fact, most of our interventions are actually aimed at correcting this imbalance, usually by increasing oxygen delivery. As a consequence, there has long been interest in the administration of blood transfusions as a way of augmenting oxygen delivery in the critically ill patient.

In recent years, a large volume of research has questioned the traditional practice of red blood cell transfusion to increase oxygen delivery. In fact, current literature suggests that there may be harm caused by significant transfusion reactions that do not include the more commonly associated, and dramatic reactions, such as hemolytic reactions, transfusion-associated lung injury, and direct infection from a pathogen contaminating a blood product. This is thought to be due to the fact that transfusions contain small amounts of donor plasma, white blood cells, antibodies, and inflammatory mediators in addition to red blood cells. A recent meta-analysis of observational studies in intensive care unit, trauma, and surgical patients found red blood cell transfusion to be an independent predictor of mortality, nosocomial infection, acute respiratory distress syndrome, and multiorgan dysfunction syndrome. The appreciation of the potential harm from transfusions has led to the use of terms such as “allogeneic blood transfusion” or even “liquid organ

transplant” to highlight the potential immunomodulating effects of transfusions.

Recent studies have greatly simplified the approach to transfusion in the nonhemorrhaging critically ill patient. In the absence of acute myocardial infarction or ischemic cerebral vascular accident (CVA), critically ill patients without specific symptoms attributable to anemia should not be transfused for hemoglobin values greater than 7 g/dL. This is best supported by randomized controlled trials comparing a hemoglobin transfusion trigger of 7 g/dL to a trigger of 10 g/dL in both a general intensive care unit population and in ICU patients with septic shock. These trials showed no benefit in using a hemoglobin transfusion trigger of 10 g/dL. As a result of these trials and increasing evidence of an association between transfusions and adverse events, most clinicians use a hemoglobin of 7 g/dL as the transfusion trigger in nonhemorrhaging ICU critically ill patients without evidence of acute ischemia.

Patients with acute coronary syndrome (ACS) or CVA have generally been excluded from most transfusion studies, because it cannot be proved that these areas of ischemia would not resolve with an increased hemoglobin level and oxygen-carrying capacity. Until more studies are completed, it is reasonable to follow current guidelines from the Society of Critical Care Medicine-Eastern Association for Surgery of Trauma, which state that transfusion may be beneficial in patients with an ACS and hemoglobin value less than 8 g/dL.

## KEY POINTS

- In asymptomatic, nonhemorrhaging critically ill patients, there is no specific hemoglobin threshold that requires a transfusion.
- For the majority of nonhemorrhaging critically ill patients, a hemoglobin of 7 g/dL should be used as the trigger for red blood cell transfusion.
- Some guidelines recommend transfusion in asymptomatic patients with a history of coronary disease once the hemoglobin level drops below 8 g/dL.
- The threshold for red blood cell transfusion in patients with ongoing ischemia is unclear; however, maintaining a hemoglobin level greater than 8 g/dL is reasonable.
- In the absence of hemorrhage, it is recommended to transfuse only one unit of red blood cells at a time.



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# PERFORM THESE SIMPLE INTERVENTIONS THAT MAKE A BIG DIFFERENCE IN PREVENTING VENTILATOR-ASSOCIATED PNEUMONIA

NICHOLAS JOHNSON, MD

Ventilator-associated pneumonia (VAP) is defined as pneumonia that occurs in a mechanically ventilated patient more than 48 hours after endotracheal intubation. VAP can occur in up to 27% of all mechanically ventilated patients and is associated with a mortality rate of almost 50%. Approximately half of all episodes of VAP occur within the first 4 days of the initiation of mechanical ventilation. Increased emergency department (ED) length of stay has been shown to be a risk factor for development of VAP. A number of simple, low-cost interventions can reduce the risk of VAP and can be implemented in the ED ([Table 42.1](#)).

**TABLE 42.1 MEASURES THAT MAY REDUCE RISK OF VENTILATOR-ASSOCIATED PNEUMONIA**

- Avoidance of endotracheal intubation when possible<sup>a</sup>
- Noninvasive modalities for oxygenation and ventilation<sup>a</sup>
- Regular assessment for liberation from mechanical ventilation<sup>a</sup>
- Semirecumbent patient positioning<sup>a</sup>
- Orotracheal and orogastric intubation as opposed to nasal<sup>a</sup>
- Selective oral decontamination<sup>a</sup>
- Avoidance of unnecessary blood transfusion<sup>a</sup>
- Application of subglottic suction<sup>a</sup>
- Antimicrobial-coated endotracheal tubes
- Endotracheal tube cuff pressure of 20–30 cm H<sub>2</sub>O<sup>a</sup>
- Gastric decompression
- Avoidance of unnecessary stress ulcer prophylaxis<sup>a</sup>
- Ventilator bundles

<sup>a</sup> Denotes measures recommended by 2005 ATS/IDSA Guidelines.

The greatest risk factors for developing VAP are endotracheal intubation and mechanical ventilation. Thus, the best strategy to prevent VAP is to avoid intubation altogether. Noninvasive positive pressure ventilation should be considered in alert patients with respiratory failure due to chronic obstructive pulmonary disease, congestive heart failure, and neuromuscular weakness. Other noninvasive ventilation modalities, such as high-flow nasal cannula, may be considered in selected patients with hypoxemia. If intubation is necessary, the orotracheal route is preferred over the nasotracheal route with regard to VAP risk.

Numerous studies have shown that the supine position facilitates aspiration of microorganisms into the lower respiratory tract. Elevating the head of the bed to 30 to 45 degrees is a simple, inexpensive intervention that likely reduces the risk for VAP. A randomized trial demonstrated a threefold reduction in VAP in patients treated in the semirecumbent position compared with patients who remained supine. Mechanically ventilated patients in the ED should be placed in the semirecumbent position unless a contraindication, such as spinal immobilization, exists.

Colonization of the oropharynx has been identified as an independent risk factor for the development of VAP. As a result, mechanically ventilated patients should receive oral decontamination. A recent Cochrane Review demonstrated a 40% decrease in the odds of developing VAP among patients treated with oral chlorhexidine. The optimal timing of this treatment remains

unclear. Two recent studies showed no benefit for oral decontamination in the prehospital setting or immediately before intubation. Whole-body chlorhexidine bathing also does not appear to decrease risk for VAP or other health care–associated infections.

Endotracheal tubes with special features may also prevent VAP. Several meta-analyses have documented a decreased prevalence of VAP when endotracheal tubes with subglottic suctioning ports were used. Numerous studies have attempted to evaluate whether antimicrobial-coated endotracheal tubes reduce VAP risk. A large randomized controlled trial demonstrated reduced incidence of VAP in patients treated with silver-coated endotracheal tubes. Subsequent analyses of the same data also showed a reduced mortality among the patients who developed VAP while intubated with silver-coated endotracheal tubes.

Attention to endotracheal tube cuff pressure may also aid in VAP prevention. ED providers should measure endotracheal tube cuff pressure after intubation. An endotracheal tube cuff pressure maintained at 20 to 30 cm H<sub>2</sub>O can prevent leakage of bacterial pathogens around the cuff into the lower respiratory tract. Devices designed to provide continuous control of endotracheal tube cuff pressure throughout the respiratory cycle have not been shown to consistently reduce risk for VAP.

Many hospitals have adopted “ventilator bundles,” based primarily on the Institute for Healthcare Improvement (IHI) initiatives to incorporate evidence-based practices into clinical care. The IHI ventilator bundle includes four practices: (1) elevation of the head of the bed to 30 to 45 degrees, (2) daily sedation vacations and assessment of readiness to extubate, (3) peptic ulcer disease prophylaxis, and (4) deep venous thrombosis prophylaxis. The impact of the IHI bundle, along with other ventilator bundles, is unclear. Nonetheless, they represent an important effort to implement best practices for VAP prevention. ED mechanical ventilation bundles that include measures such as arterial blood gas sampling, gastric decompression, early sedation, appropriate initial tidal volume, and quantitative capnography have been shown to improve outcome; however, the role of these bundles strictly in VAP prevention remains uncertain.

## KEY POINTS

- The best way to avoid VAP is to avoid unnecessary intubation and mechanical ventilation.
- Elevation of the head of the bed and selective oral decontamination

may reduce VAP risk.

- Endotracheal tubes with subglottic suctioning ports or antibiotic coating have been shown to decrease the prevalence of VAP.
- Maintaining endotracheal tube cuff pressure at 20 to 30 cm H<sub>2</sub>O can prevent leakage of bacteria into the lower respiratory tract.
- Ventilator bundles that have been adapted for the ED may improve patient outcomes.

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# **KNOW HOW TO CARE FOR THE ICU BORDER IN YOUR ED**

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**JOSHUA D. FARKAS, MD, MS**

The volume of critically ill patients presenting to the emergency department (ED) is rapidly growing. In addition to an increasing number, a prolonged length of stay can significantly impact the mortality of critically ill ED patients. The value of high-quality supportive care cannot be overstated, and it should be initiated in the ED while awaiting transfer to the intensive care unit (ICU).

## **AVOID EXCESS FLUID ADMINISTRATION**

Critically ill patients can gain ~1 L of fluid per day due to a myriad of sources. Multiple intravenous medications, continuous infusions, and repeated fluid boluses can lead to an excessively positive fluid balance. Over several days, this excess fluid may cause severe edema and lead to severe complications, including pulmonary edema, renal failure, abdominal compartment syndrome, and pressure ulceration.

Avoiding unnecessary fluid administration is critical. Reflexively giving fluid boluses to increase blood pressure or urine output should be avoided unless the patient is truly volume depleted. Among patients with systemic inflammation (e.g., sepsis, pancreatitis), fluid boluses may cause temporary improvement, but this fluid rapidly extravasates into the tissues. In addition to indiscriminate fluid boluses, “maintenance fluids” should also be avoided, as this can lead to excessive fluid administration.

## **AVOID UNNECESSARY BLOOD TRANSFUSION**

Blood transfusion can cause several complications, including volume overload, immunosuppression, transfusion reactions, and transfusion-related acute lung injury. Nonbleeding ICU patients generally should not be transfused unless they have a hemoglobin <7 mg/dL. Transfusing two units of blood at a time should also be avoided, as a patient's hemoglobin level will fluctuate over time. A recent trial investigating upper gastrointestinal hemorrhage showed that a hemoglobin threshold of 9 mg/dL increased mortality compared to a hemoglobin threshold of 7 mg/dL for blood transfusion. Importantly, this study excluded patients with exsanguinating hemorrhage or acute coronary syndrome.

## **AVOID OVERSEDATION AND UNDERTREATMENT OF PAIN IN THE INTUBATED PATIENT**

Once the acute resuscitation phase has ended, the goal for intubated patients is to be awake and comfortable on the ventilator. Compared to deep sedation, lighter levels of sedation reduce delirium and the duration of intubation. The first step toward rendering a patient comfortable on a ventilator is the treatment of pain. The majority of intubated patients experience pain. A common error in pain management of the critically ill is to simply provide a sedative. Most sedative medications provide no analgesia and will not provide adequate comfort until the dose is increased to near coma-inducing levels. The key to achieving an awake and comfortable state typically starts with an opioid (e.g., fentanyl 25 to 150 mcg/h), which will relieve pain while providing some sedation. Opioids are the recommended first-line agents for analgesia in the critically ill.

Often, patients will also require a sedative medication. Although benzodiazepines have traditionally been used, recent evidence shows that they increase the risk of delirium and duration of mechanical ventilation. The primary advantage of benzodiazepines is that they cause less hypotension than do other sedatives, so they may remain useful in refractory shock.

Propofol is a frequently used sedative. High doses of propofol may cause hypotension and increase the risk of propofol infusion syndrome. These complications may be avoided by combining a low-dose propofol infusion (e.g., up to 30 mcg/kg/min) with a moderate-dose opioid.

For patients with persistent agitation despite an opioid and sedative, atypical antipsychotics may be a helpful adjunctive therapy. More sedating agents (e.g., quetiapine and olanzapine) are generally used at night to reduce sleep-wake cycle disturbance.

Dexmedetomidine is another sedative option. The primary advantage of dexmedetomidine is that it does not suppress respirations, so that it may be used to bridge the patient through the extubation process (thus avoiding pre-extubation anxiety and tachypnea). Drawbacks of dexmedetomidine include cost, hemodynamic fluctuations, and a risk of tachyphylaxis and withdrawal if used at high doses for more than 4 to 5 days. Overall, dexmedetomidine is not frequently used as an initial sedative for intubated patients in the ED.

## **AVOID NONSTEROIDAL ANTI-INFLAMMATORY MEDICATIONS**

Nonsteroidal anti-inflammatory medications (NSAIDs) are a commonly used medication for the treatment of pain and inflammation in ambulatory patients, but in the critically ill, these drugs can have devastating consequences. NSAIDs increase the risk of renal failure and increase the risk of stress ulceration. It is generally best to avoid these unless a specific indication exists (e.g., pericarditis).

## **AVOID DEEP VEIN THROMBOSIS**

Patients should receive deep vein thrombosis (DVT) prophylaxis unless they are actively bleeding or have other contraindications. Common approaches include unfractionated heparin (e.g., 5,000 IU every 8 hours) or low molecular weight heparin (e.g., enoxaparin 40 mg daily). In renal failure, low molecular weight heparin is contraindicated. Recall that heparin is *weight based*, so morbidly obese patients need proportionally higher doses (e.g., 0.25 mg/kg enoxaparin every 12 hours).

### **KEY POINTS**

- Avoid unnecessary maintenance fluids.
- Don't transfuse unless the patient is actively exsanguinating or the hemoglobin is <7 mg/dL.
- Treat pain in mechanically ventilated patients.
- Avoid benzodiazepines and NSAIDs if possible.
- Provide DVT prophylaxis unless contraindicated.



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# KNOW HOW TO EVALUATE AND MANAGE THE INTUBATED PATIENT WITH REFRACTORY HYPOXEMIA

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Refractory hypoxemia in an intubated patient is a critical emergency that demands a rapid, systematic approach. Assessment and management are performed in unison, with life threats addressed in order of priority. The “DOPEs” mnemonic ([Table 44.1](#)) can be used to guide initial actions, especially in the immediate postintubation period.

**TABLE 44.1 THE “DOPEs” MNEMONIC**

- Displaced ETT
- Obstructed ETT
- Patient disorders, such as pneumothorax
- Equipment problems
- “Stacked breaths” (dynamic hyperinflation)

The first step in the management of the intubated patient with refractory

hypoxemia is to disconnect the ventilator from the endotracheal tube (ETT). Once disconnected, look for signs of gas trapping (AKA auto-PEEP or dynamic hyperinflation). If gas trapping is suspected, allow for prolonged expiration by adjusting the ventilator settings. This can be accomplished with a lower respiratory rate (e.g., 6 breaths/min), increased inspiratory flow rate (e.g., 100 L/min), prolonged expiratory time (e.g., I:E ratio of 1:4 or more), or decreased PEEP (e.g., PEEP = 0 cm H<sub>2</sub>O).

Once dynamic hyperinflation is excluded, connect a bag-valve device to the ETT with 100% oxygen. If the patient is easy to ventilate and reoxygenate, then the culprit is either the ventilator or the circuit. If the patient is still difficult to ventilate, then there is a problem with the ETT or the patient. Importantly, never ventilate a patient with a tracheostomy before confirming tube position and patency, due to the risk of causing catastrophic subcutaneous emphysema.

Confirm ETT patency by monitoring end tidal carbon dioxide (ETCO<sub>2</sub>) and passing a suction catheter. Quantitative ETCO<sub>2</sub> monitoring is the gold standard for confirmation of endotracheal intubation and for detection of ETT malposition. Recall that up to eight ETCO<sub>2</sub> waveforms over 30 seconds may be seen after esophageal intubation. If a bougie is used to confirm ETT position and patency, it should be passed gently to avoid tracheobronchial injury. “Hold up” of the bougie occurs at the carina (~30 cm) when the ETT is endotracheal. With esophageal ETT placement, the bougie will pass easily beyond 35 cm. Bronchoscopy or chest radiography can also be used to confirm ETT position; however, these modalities may take too long in the rapidly deteriorating patient.

Patients who are easy to ventilate with the bag-valve device and yet remain hypoxemic may have a malpositioned ETT or a circuit leak (e.g., cuff leak, disconnection, or a breach in the circuit). If in doubt, remove the ETT, continue bag-valve-mask ventilation, and prepare for reintubation.

If the above etiologies have been excluded and the patient remains hypoxemic, consider patient factors. To rapidly assess patient factors, use the “MASH” approach during bag-valve ventilation ([Table 44.2](#)). Asymmetric chest movement raises suspicion for pneumothorax, pleural effusion, hemothorax, or lung collapse due to pneumonia. Other common patient causes of hypoxemia include pulmonary edema, bronchospasm, and pulmonary embolus.

|   |
|---|
| <b>TABLE 44.2 THE “MASH” APPROACH FOR RAPID PATIENT</b> |
|---|

## ASSESSMENT

- Movement of the chest
  - Check for bilateral, unilateral, or absent chest movement.
  - Is the chest hyperexpanded?
- Arterial saturation (SaO<sub>2</sub>) and PaO<sub>2</sub>
  - Obtain an arterial blood gas sample if time permits
- Skin color of the patient (check for cyanosis)
  - Remember that the SpO<sub>2</sub> monitor lags behind the patient's true oxygen saturation
- Hemodynamic stability

Suspect acute respiratory distress syndrome (ARDS) in patients with bilateral pulmonary infiltrates on chest radiography. These patients require protective lung ventilation with tidal volumes of  $\leq 6$  mL/kg predicted body weight, plateau pressures  $< 30$  cm H<sub>2</sub>O, and an arterial pH above 7.15 (allowing for “permissive hypercapnoea”). Avoid excess fluid administration and perform head up positioning to help preserve functional residual capacity. Useful therapies for refractory hypoxemia in ARDS are listed in [Table 44.3](#).

### TABLE 44.3 THERAPIES FOR THE ARDS PATIENT WITH REFRACTORY HYPOXEMIA

- Administer sedation and neuromuscular blockade to optimize patient-ventilator synchrony.
- Provide suction, chest physiotherapy, and bronchoscopy to shift sputum plugs.
- Ventilator adjustments:
  - Optimize PEEP using the ARDSNet nomogram. Additional PEEP may be required in the obese patient.
- Increase the inspiratory time (and consequently the I:E ratio).
- Inverse ratio ventilation or alternate ventilation modes, such as airway pressure release ventilation, can be tried if the clinician has sufficient expertise. No particular strategy is proven to improve patient outcomes.
- Recruitment maneuvers: No strategy is proven superior; a simple approach is to apply 40 cm H<sub>2</sub>O of positive pressure for 40 s.
- Prone positioning: Can be performed in settings with teams trained in the procedure. Prolonged proning (e.g., 16 hours per day) is associated with decreased mortality in severe ARDS.
- Extracorporeal membrane oxygenation: Venovenous extracorporeal membrane oxygenation is used for reversible respiratory disorders resulting in hypoxemia refractory to other measures.

Nonpulmonary causes of hypoxemia (e.g., cyanotic heart disease or

hemoglobinopathies) are rare but should be considered in refractory cases. Finally, if the patient is easy to ventilate and the hypoxemia rapidly resolves, establish the immediate antecedents to hypoxemia. Simple disconnection of the ventilator/circuit or suctioning can lead to significant desaturation through derecruitment and atelectasis (especially in small children).

## KEY POINTS

- Use the “DOPEs” mnemonic to evaluate immediate life-threatening complications in the intubated patient with hypoxemia.
- Disconnection from the ventilator can treat dynamic hyperinflation and excludes equipment causes.
- Quantitative ETCO<sub>2</sub> monitoring is the gold standard for confirming ETT positioning.
- The “MASH” approach allows rapid assessment of the patient while attempting to diagnose causes of hypoxemia.
- Numerous ventilatory and nonventilatory strategies can help improve refractory hypoxemia in patients with ARDS.

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# **READY FOR PRIME TIME? EXTRACORPOREAL LIFE SUPPORT IN THE ED**

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Cardiac arrest is a complex event in the emergency department (ED). Termination of resuscitation (TOR) guidelines have pushed prehospital systems to stay on the scene rather than transport to the hospital. This has resulted in fewer ED presentations and more prolonged arrests in those who do arrive. The survivorship of out-of-hospital cardiac arrest (OHCA) and in-ED cardiac arrest is <10% but improved by a strategic and organized approach to resuscitation. Two of the most important components to survivorship of cardiac arrest are high-quality chest compressions and early defibrillation of ventricular tachydysrhythmias. The goal of chest compressions is to increase the coronary perfusion pressure (CPP) above 25 mm Hg, which has been associated with substantial increases in return of spontaneous circulation (ROSC). Achieving high-quality chest compressions and a CPP above 25 mm Hg is a difficult task and involves minimizing interruptions, coordinated transitions of chest compression providers, and a focus on quality chest compressions around defibrillation. Mechanical chest compression devices may improve these metrics, but literature supporting superior efficacy of these devices is lacking.

Assessing the quality of chest compressions has been aided by recent advances in end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>) monitoring along with devices that provide real-time feedback to providers. ETCO<sub>2</sub> has additionally aided practitioners in determining ROSC during the cardiac arrest. Increase in CO<sub>2</sub> exuded through the lung suggests spontaneous or artificial generation of

blood flow and therefore can be used to judge quality of chest compressions and ROSC. Recent literature using near-infrared spectroscopy suggests not only benefit in assessing ROSC but potential use in neuroprognostication. A tool that can accurately assess a patient's capacity for neurologic recovery during a cardiac arrest would be a monumental discovery. If a patient was deemed neurologically salvageable, many advanced resuscitation tools would see greater use.

One technology that has offered the potential for substantial increases in the survivorship of cardiac arrest is extracorporeal life support (ECLS). ECLS involves bypassing the native heart and lungs by providing these functions outside of the body. In a cardiac arrest, ancillary support of perfusion, oxygenation, and ventilation can allow for far superior CPP compared to traditional chest compressions. Many of the limitations of conventional CPR can be minimized with mechanical circulatory support provided by an extracorporeal cardiopulmonary bypass circuit. A common mistake in ECLS is failure to realize your hospital already has this technology. Discussion with your cardiothoracic surgeons may reveal the ability to use ECLS without the substantial efforts needed to organize a new program.

For institutions with ECLS capability, the appropriate patient selection can be challenging. This decision is based on our currently imprecise ability to provide accurate neuroprognostication in the arrested patient. Historical characteristics that favor good neurologic outcome include short arrest time, initial rhythm of ventricular tachycardia or ventricular fibrillation, bystander cardiopulmonary resuscitation, and younger age. Special circumstances including the hypothermic patient and patient who presents in cardiac arrest as a result of toxic ingestion show particular promise with the use of ECLS. While ECLS is a heroic effort in the salvage of patients in refractory cardiac arrest, the benefits can be dramatic.

Complications associated with ECLS can be significant. These include retroperitoneal placement of a catheter, venous or arterial laceration, pulmonary edema associated with insufficient right atrial drainage or aortic insufficiency, leg ischemia from inadequate perfusion distal to the cannulated femoral artery, and hemorrhage secondary to induced and acquired coagulopathies. Common mistakes involve unintentional clamping of the extracorporeal circuit, failing to attach oxygen to the pump, attempting to increase oxygenation by increasing oxygen supply, and failing to get a right radial arterial blood gas after initiation of bypass.

Good neurologic outcome following cardiac arrest is infrequent; so it is imperative that we continue to search for different strategies to improve these

outcomes. The utilization of ECLS in the ED is a new and exciting field. Currently, there are a number of unanswered questions regarding who could potentially benefit from this technology; however, early reports of the potential benefit are promising and should encourage further investigation.

## KEY POINTS

- Determine if your institution has the capability to perform ECLS.
- When establishing an ED ECLS program, it is imperative that there is a strong, collaborative relationship between emergency medicine, critical care, cardiology, and cardiothoracic surgery.
- Initiating ECLS during cardiac arrest can be challenging, so it is imperative that extensive technical, procedural, and systems training occur before attempting in a clinical setting.
- When initiating ECLS, clearly identified roles need to be assigned prior to the procedure being performed.
- An in-depth understanding of the limitations, contraindications, and complications associated with ECLS is critical when considering its implementation in the ED.

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# **RAPIDLY REVERSE LIFE-THREATENING HEMORRHAGE IN THE PATIENT TAKING AN ORAL ANTICOAGULANT MEDICATION**

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**RORY SPIEGEL, MD**

Oral anticoagulant medications are commonly prescribed. Whether it is a traditional vitamin K antagonist (VKA) or a novel oral anticoagulant (NOAC) medication, all carry the risk of serious hemorrhage.

## **TRADITIONAL VITAMIN K ANTAGONISTS**

VKAs inhibit vitamin K epoxide reductase, which prevents the formation of vitamin K–dependent coagulation factors II, VII, IX, and X. The most ubiquitous test to assess anticoagulation in patients on VKAs are prothrombin time (PT) and international normalized ratio (INR). Vitamin K–dependent factor levels must decrease to 20% to 30% of normal values before changes in INR are consistently observed. The most common VKA is warfarin.

Current recommendations for VKA reversal are listed in [Table 46.1](#). For the nonbleeding patient with an INR > 10, vitamin K should be administered. Available routes of administration include oral, subcutaneous, intramuscular, and intravenous (IV). Though IV administration corrects the INR slightly faster than does the oral route, differences in reversal time are rarely clinically significant. Due to erratic efficacy and risk of intramuscular hematoma formation, subcutaneous and intramuscular routes are not

recommended.

**TABLE 46.1 RECOMMENDED INTERVENTIONS FOR AN ELEVATED INR**

|   |   |
|---|---|
| 3 < INR < 5 with no significant bleeding  | Lower or omit next warfarin dose  |
| 5 < INR < 10 with no significant bleeding | Omit next 1–2 doses of warfarin   |
| INR > 10 with no significant bleeding     | Hold warfarin therapy<br>Give 2.5–5 mg of vitamin K PO  |
| Serious bleeding with any elevated INR    | Hold warfarin therapy<br>Give 10 mg of vitamin K IV<br>Give 25–50 U/kg PCC or 10–15 mL/kg FFP |

For the bleeding patient with any elevated INR, fresh frozen plasma (FFP) should be rapidly administered. FFP directly replaces all vitamin K–dependent factors in a dose-dependent fashion. A dose of 10 to 15 mL/kg is recommended to replace 25% of factor levels. Prothrombin complex concentrate (PCC) is a concentrated form of vitamin K–dependent factors and is available as 3- or 4-factor products. The 3-factor products contain II, IX, and X, whereas 4-factor PCCs contain II, VII, IX, and X. PCCs deliver a large quantity of factors in a low volume and have fewer side effects when compared with FFP. Current recommended doses for PCCs range between 25 and 50 U/kg. For 3-factor products, some have recommended the addition of activated factor VII to compensate for the insufficient quantities of factor VII. Currently, the American College of Chest Physicians recommends 4-factor PCCs over FFP in the treatment of life-threatening hemorrhage due to VKAs. To date, trials comparing the efficacy of PCCs to FFP have shown significantly faster correction of INR with PCCs, but have failed to find a significant difference in mortality.

## NOVEL ORAL ANTICOAGULANT MEDICATIONS

NOACs have recently become an alternative to VKAs. Current products include direct thrombin inhibitors (i.e., dabigatran) and factor Xa inhibitors (i.e., rivaroxaban, apixaban, edoxaban). These products do not deplete factors like VKAs; rather, they cause active inhibition. In pharmaceutical-sponsored trials, NOACs were shown to be noninferior to warfarin in both efficacy and risk profile. Traditional anticoagulation tests (i.e., PT, PTT, INR) are inaccurate, insensitive, and unable to quantify the degree of anticoagulation in patients taking an NOAC. Instead, a thrombin time or

ecarin clotting time is recommended to evaluate direct thrombin inhibition, whereas anti-factor Xa levels should be obtained when factor Xa inhibition is suspected. Most NOACs have short half-lives, with anticoagulative effects resolving within 24 hours of the last dose.

Current guidelines recommend the use of 4-factor PCCs, activated factor VII, or factor VIII inhibitor bypassing activity to treat hemorrhage from NOACs. Recent trials have demonstrated variable success when using these products to reverse the effects of NOACs. In contrast to Xa inhibitors, dabigatran can be partially cleared with hemodialysis. Volunteer studies and case reports have demonstrated that up to 68% of circulating dabigatran can be dialyzed in a single 2- to 4-hour session. The clinical presentation and the degree of blood loss will determine the viability of dialysis as a treatment option.

The Food and Drug Administration recently approved reversal agents for both dabigatran and the Xa inhibitors. Idarucizumab is a monoclonal antibody fragment that binds dabigatran in the serum and neutralizes its effect. Andexanet is a compound that binds to the site of anti-Xa inhibitors, thereby preventing further inhibition of Xa. To date, trials evaluating these products are industry sponsored and require further independent study. Nonetheless, their use should be considered in the patient with life-threatening hemorrhage due to an NOAC medication.

## KEY POINTS

- Vitamin K is not recommended in patients on warfarin with no active bleeding and an INR < 10.
- The initial dose of FFP for the hemorrhaging patient on warfarin is 10 to 15 mL/kg.
- 4-Factor PCCs will rapidly correct the INR but have not been shown to improve mortality when compared to FFP.
- Dabigatran can potentially be dialyzed in the event of life-threatening hemorrhage or overdose.
- Consider idarucizumab for emergent reversal of hemorrhage due to dabigatran.

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# **BE READY TO DISCUSS AND DELIVER END-OF-LIFE CARE IN THE EMERGENCY DEPARTMENT**

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**ASHLEY SHREVES, MD**

Patients commonly visit the emergency department (ED) near the end of life (EOL). The needs of dying patients can easily overwhelm caregivers, even in the presence of hospice services. Inpatient palliative care resources are increasingly available, but not able to meet the large demand generated by such patients in the ED. It is critical that emergency providers be ready to discuss and deliver EOL care.

For some dying patients, EOL decision-making has already occurred and been recorded within advance directives. Be sure to ask if EOL decisions have been made or if documentation exists regarding EOL decisions. It is critical to ask about such documents especially in patients presenting without decision-making capacity. Importantly, the Physician Orders for Life-Sustaining Treatment (POLST) addresses the most common interventions considered at the EOL and can provide practical guidance for care in the ED. Emergency providers have expressed concern about their legal jeopardy should they withhold treatments in accordance with the POLST. The POLST has been used successfully for over a decade in some states, and there has not been a single malpractice case generated surrounding its use.

In the absence of advance directives, directly engage patients and their surrogate decision makers to determine the optimal care pathway. A common error is to give patients and families a menu of treatment options. This can lead to medical decision-making that is driven by poor health literacy and overwhelming fear of the consequences of their decisions. Elicit the patient's

values and goals and then use that information to make treatment recommendations.

Conversations surrounding EOL care go best when performed in a systematic fashion. Most available “goals of care” pathways share the following common steps:

- 1) Preparation
- 2) Patient perception
- 3) Invitation
- 4) Knowledge exchange
- 5) Elicit values/goals
- 6) Medical recommendation

For step 1, ensure that you have an accurate understanding of the clinical facts. This may necessitate a discussion with the patient’s primary care provider or oncologist. For step 2, explore the patient’s perception of his or her illness. Phrases like “What have the doctors told you about your illness?” and “What’s your sense of how things are going?” are useful. For step 3, assess the patient/family readiness to proceed through what is likely to be an emotionally painful conversation. Use phrases like “Would it be ok if I shared my understanding of this situation?” or “I’m worried that I have bad news about your disease, would it be ok if we discussed this right now?” Including this step ensures that the patient and family are in control of the medical information that they receive.

Next, share your understanding of the clinical situation. Share information in small bites and avoid medical jargon, when possible. Use phrases like “I think she’s dying” and “time could be short” if consistent with the clinical picture. Phrases like “I’m really worried” can be more effective at communicating a poor prognosis than statistics.

For step 5, explore what is most important to patients and their caregivers. Some will respond well to open-ended questions like “On hearing this news, what’s most important to you?” and “Can you help me understand what is an acceptable quality of life to you?” Others will need more concrete guidance. For instance, “Some people in your situation would be willing to be put on life support and spend time in the ICU, even if there was only a small chance it would buy them more time or get them home with their family again. Other people would be unwilling to go through that and would prefer that we refocus on their comfort at this point. What kind of person are you?”

In step 6, discourage the use of life-prolonging treatments if they are inconsistent with the patient’s goals. For example, “I’m worried that using

treatments like life support will only prolong her dying and add to her suffering” or “The best thing we can do right now is refocus all our efforts on her comfort and quality of life.” Brief, direct statements like “In light of what we’ve discussed, it is in your loved one’s best interest to have a natural death” and “When her heart stops, we will not interfere with that process” can address code status. Detailed descriptions of chest compressions and defibrillation are rarely necessary.

Clinicians should expect strong emotions in response to hearing bad news. Facing death can trigger feelings of grief, despair, anger, guilt, disappointment, shock, and more. This is normal and does not reflect poorly on the clinician’s communication skills. Rather than saying “It’s going to be ok,” use phrases like “I wish things were different” and “I can only imagine how disappointed you must be” to show empathy.

## KEY POINTS

- Emergency providers must learn how to have EOL conversations.
- EOL conversations go best when done systematically.
- Patients at the EOL should never be given a menu of treatment options and be expected to choose wisely.
- Physicians should recommend treatments that meet patient goals.
- EOL conversations provoke strong emotional responses from patients and families. This is normal and does not reflect poorly on the clinician’s communication skills.

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# **SECTION III**

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# **CARDIOLOGY**



# **RECOGNIZE ATYPICAL PRESENTATIONS OF ACUTE CORONARY SYNDROME**

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**AMITA SUDHIR, MD**

The classic presentation of an acute coronary syndrome (ACS) is that of an older age male smoker with hypertension who reports exertional chest pressure that radiates to the left upper extremity and is associated with shortness of breath. This presentation is well recognized by the emergency provider (EP). Unfortunately, many patients with ACS lack overt chest pain or pressure and present to the EP with atypical signs and symptoms. As a result, the EP may fail to consider the diagnosis of ACS, leading to increased morbidity and mortality. Recent literature has demonstrated that patients with atypical ACS presentations are less likely to receive anti-ischemic therapy and more likely to die when compared to patients with the classic ACS presentation. Currently, more than 20% of ACS patients who present with atypical signs and symptoms are missed upon initial evaluation. In order to prevent unnecessary morbidity and mortality, it is critical for the EP to identify patients with ACS who do not present with textbook symptoms.

Common atypical ACS symptoms include dyspnea, diaphoresis, nausea, vomiting, and near-syncope. Jaw pain, neck pain, back pain, extremity pain, abdominal pain, and fatigue are additional symptoms that can be caused by an ACS. Due to the absence of chest pain with these symptoms, an electrocardiogram (ECG) and cardiac markers are often delayed or not obtained altogether.

Patients with atypical ACS presentations are more likely to be female or elderly or have a history of diabetes or heart failure. Women are at

particularly high risk for misdiagnosis of ACS. Not only are women more likely to have atypical presentations, they are also more likely to have diabetes and be younger at the time of ACS presentation. In addition, women present later in the time course of their illness when compared to ACS patients who present with the classic symptom of chest pain. Patients with diabetes have long been recognized to present with atypical features of ACS. This higher rate of atypical ACS presentation is thought to be due to a neuropathy that affects the sensory innervation to the heart. Finally, patients with atypical ACS presentations are less likely to have a history of smoking, hyperlipidemia, or prior cardiac disease. Importantly, concern for ACS should not solely be limited to female, diabetic, or older patients who present with atypical symptoms. ACS should be considered for any symptom without any obvious cause that could potentially be caused by cardiac ischemia.

The most important step in making the diagnosis of ACS is to simply consider the diagnosis in patients presenting with the symptoms listed above. Obtain an ECG in any patient that there is clinical concern for ACS. If the ECG is nondiagnostic, consider obtaining cardiac markers and placing patients in observation for additional diagnostic testing. Furthermore, it is important to administer time-sensitive therapy when the diagnosis of ACS is considered. This may include antiplatelet or anticoagulant medications, along with cardiology consultation.

## KEY POINTS

- Patients with atypical ACS presentations are less likely to receive critical, time-dependent therapy and are more likely to die.
- Dyspnea, diaphoresis, vomiting, and fatigue are common atypical signs and symptoms of ACS.
- Atypical presentations of ACS are more likely to occur in women, diabetic patients, and the elderly.
- Patients with atypical ACS presentations are less likely to have existing cardiac disease.
- Obtain an ECG in any patient with signs or symptoms concerning for ACS.

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# **TYPE A BEHAVIOR: CONSIDER AORTIC DISSECTION IN PATIENTS WITH CHEST PAIN AND ISCHEMIC ELECTROCARDIOGRAMS**

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**JESSICA BALDERSTON, MD AND  
JEFFREY D. FERGUSON, MD, FACEP, NRP**

In the hectic environment of the emergency department, rapid diagnosis and emergent management of life-threatening conditions must occur. In the patient with acute chest pain and an electrocardiogram (ECG) concerning for an ST-segment elevation myocardial infarction (STEMI), the diagnosis and treatment seem straightforward. National guidelines recommend emergent reperfusion with percutaneous coronary intervention (PCI) or fibrinolytic therapy. Unfortunately, the potential for patient harm is high when an acute aortic dissection (AD) causes an acute coronary syndrome (ACS) and produces ischemic ECG changes.

In AD, a defect in the aortic intima allows entry of blood into the tunica media. A false lumen develops and can propagate in an anterograde or retrograde direction. ADs that involve the ascending aorta (Stanford type A) can cause coronary insufficiency by several mechanisms. These include a circumferential intimal defect of the ascending aorta that drapes over the coronary ostia, an intimal defect involving the coronary ostia itself, propagation of the false lumen into the coronary vessel that compresses the true lumen, or circumferential intimal detachment within the coronary artery causing intussusception of the inner vessel wall. The right coronary artery is the most common vessel affected by these pathologic mechanisms. As a

result, an inferior STEMI is the most common ACS presentation in patients with AD. Notwithstanding, case reports exist of acute AD with coincident STEMI without the dissection directly involving the coronary vessel (e.g., STEMI with Stanford type B).

Approximately 70% of patients with acute AD will have an ECG abnormality. These can include nonspecific ST-segment or T-wave changes, left ventricular hypertrophy, and atrial dysrhythmias. Diffuse ST-segment elevation, with or without PR segment depression, and electrical alternans should raise suspicion for pericardial effusion caused by aortic rupture into the pericardial space. While the rate of AD in patients with STEMI is very low (0.1% to 0.2%), the rate of STEMI is 5% in patients with an AD. This rate rises to 8% when considering only those patients with a type A dissection.

Consider the diagnosis of AD, especially in patients with an inferior STEMI. Additional history and physical examination findings may prevent catastrophic results from antiplatelet, anticoagulant, or fibrinolytic therapy. Importantly, an anterior STEMI is rarely, if ever, associated with an acute AD.

How should the clinician approach the patient with acute chest pain, ischemic ECG findings, and risk factors for an acute AD? A presentation in which acute AD is strongly suggested and an inferior or posterior STEMI is noted on ECG should prompt rapid evaluation of the thoracic aortic. Fibrinolytic therapy should be withheld until AD can be excluded. Confirmation or exclusion of AD can be rapidly accomplished with computed tomography of the chest and abdomen. Emergent PCI can also establish the diagnosis in select cases. When the clinical presentation is concerning for an acute AD and nondiagnostic ECG abnormalities are seen, a similar diagnostic-therapeutic approach is warranted.

## KEY POINTS

- Up to 70% of patients with acute AD will have an ECG abnormality.
- Up to 8% of type A dissections can be complicated by STEMI.
- The right coronary artery is the most common artery involved in cases of AD.
- An anterior STEMI is rarely associated with an acute AD.
- Do not administer fibrinolytic therapy to STEMI patients in whom there is suspicion of an acute AD.

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# **CONTENTS UNDER PRESSURE: AGGRESSIVE HEMODYNAMIC MANAGEMENT IN PATIENTS WITH ACUTE AORTIC DISSECTION**

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**JEFFREY D. FERGUSON, MD, FACEP, NRP AND  
JESSICA BALDERSTON, MD**

Acute aortic dissection (AD) is defined as a tear through the intima, which results in the flow of arterial blood between the layers of the aorta. This intimal tear is usually preceded by degeneration of the medial layer of the aortic wall that is caused by various disease processes that include atherosclerosis, congenital weakness of structural components, or vasculitis. Regardless of the pathologic processes, the medial layer is weakened and can tear with any increase in aortic wall stress. The most common system used to classify acute ADs is the Stanford classification system. Any dissection involving the ascending aorta is classified as Stanford type A, whereas Stanford type B is limited to dissections involving the descending aorta. More recently, the term intramural hematoma (IMH) has been used to characterize a thrombus within the wall of the aorta that does not enhance with the administration of contrast agents for computed tomography or magnetic resonance imaging. Since the clinical presentation and progression of IMH is similar to an acute AD, management of both conditions is identical. Regardless of the presence of an acute AD or IMH, hemodynamic monitoring in multiple limbs may be needed to obtain an accurate blood pressure measurement and guide therapy.

The foundation of AD management is based on the theory that a

reduction in sheer stress on the aortic wall will slow propagation of the dissection and prevent either aortic rupture or compromised flow to vital organs. The therapeutic targets to reduce sheer stress are blood pressure and heart rate (HR). Classic teaching is to reduce the HR to ~60 beats per minute (bpm), followed by a rapid reduction in systolic blood pressure (SBP) to <120 mm Hg. Though recent consensus guidelines continue to recommend these targets for HR and SBP, it is important to note that these numbers have not been studied in prospective or randomized trials. In fact, there is only scant observational evidence that reducing HR has any effect on mortality. To that end, current European guidelines for the treatment of AD focus on blood pressure reduction alone. The most reasonable approach to managing patients with acute AD is to lower SBP and HR as much as tolerable while maintaining perfusion to vital organs. Therapy should be individualized and based on markers of end organ perfusion, such as mentation and urine output.

Beta-blocker medications are recommended as the initial agent in acute AD. This will lower HR as well as blunt potential reflex tachycardia commonly seen with vasodilatory medications. Esmolol and labetalol are the two most common beta-blocker medications used. Both can be administered as continuous infusions. Esmolol has the advantage of a shorter half-life compared with labetalol, should the infusion need to be discontinued due to hemodynamic compromise. Importantly, beta-blockers should be avoided in patients with acute aortic insufficiency (i.e., new diastolic murmur, pulmonary edema, tachycardia), as they may cause cardiovascular collapse. For patients in whom beta-blockers are contraindicated or poorly tolerated, a calcium channel blocker (e.g., diltiazem) can be used. Following a reduction in HR, vasodilatory agents (e.g., nicardipine) should be administered to reduce SBP. Do not forget to administer an analgesic medication to patients with an acute AD. Pain contributes to elevation of HR and SBP and should be managed. Intravenous opioids (e.g., fentanyl) are recommended.

Immediate surgical consultation should be obtained for Stanford type A dissections, complicated Stanford type B dissections and those at high risk for impending aortic rupture. Complicated Stanford type B dissections are those that demonstrate end organ ischemia to organs such as the spine, kidney, and splanchnic circulation. Stanford type B dissections that do not have evidence of end organ ischemia or impending rupture are generally managed medically with control of blood pressure and HR.

For patients with an acute AD complicated by cardiac tamponade, pericardiocentesis should be performed only as a last resort. If necessary, remove only enough blood to restore adequate circulation while preparing to move the patient to the operating room. Additional fluid removal may increase flow and propagation of the false lumen.



## KEY POINTS

- Current guidelines recommend rapidly reducing HR to approximately 60 bpm followed by a reduction in SBP to 120 mm Hg.
- Beta-blocker medications, such as esmolol or labetalol, are the initial recommended agents to reduce HR.
- Administer a vasodilator agent once HR is controlled.
- Administer an analgesic, such as fentanyl, to all patients with an acute AD.
- Stanford type A, complicated Stanford type B, and those with signs of impending rupture should receive emergent operative therapy.

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# DO NOT CONFUSE MULTIFOCAL ATRIAL TACHYCARDIA WITH ATRIAL FIBRILLATION

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CHRISTOPHER GREENE, MD

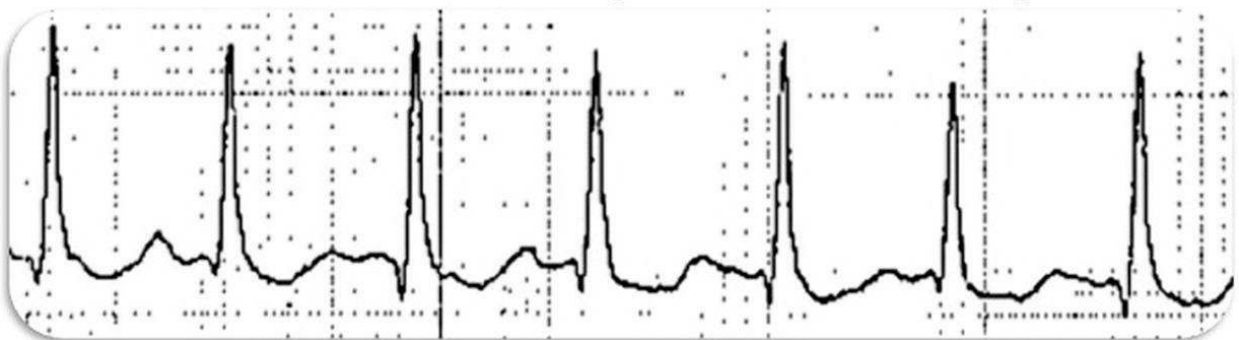
Multifocal atrial tachycardia (MAT) is a rare, poorly understood, and commonly misdiagnosed dysrhythmia. The overall prevalence of MAT is <0.4%. When present, it is usually seen in acutely ill patients, such as those with hypoxia, hypercarbia, hypokalemia, hypomagnesemia, or hyperadrenergic states, or in patients with acute exacerbations of chronic disease. In fact, it is felt that MAT reflects the severity of the underlying disease process. The most common misdiagnoses of MAT are atrial fibrillation (Afib) and sinus tachycardia. Since the treatment of each of these dysrhythmias is different, it is imperative for the emergency provider to correctly diagnose MAT.

Differentiating between MAT and Afib ([Figure 51.1](#)) can be a challenge for providers. Both dysrhythmias are irregularly irregular, can cause rapid ventricular rates, and more commonly occur in elderly patients with existing cardiopulmonary disease. In addition, MAT can occasionally be associated with other atrial dysrhythmias, including Afib and atrial flutter (AF). The most commonly accepted diagnostic criteria for MAT are (1) the presence of at least three morphologically distinct P waves in a single electrocardiogram (ECG) lead, (2) an atrial rate greater than or equal to 100 beats per minute, and (3) an isoelectric baseline between P waves. P waves in MAT are well formed and discrete, compared to Afib where P waves are characterized as continuous, undulating, and changing. Additional features include the absence of a dominant pacemaker and irregularly irregular P-P, P-R, and R-R intervals. [Table 51.1](#) lists key features of MAT, Afib, and AF.

### Multifocal Tachycardia



### Atrial Fibrillation with Rapid Ventricular Response



**Figure 51.1** Multifocal atrial tachycardia and atrial fibrillation.

**TABLE 51.1 COMPARISON OF MULTIFOCAL ATRIAL TACHYCARDIA, ATRIAL FIBRILLATION, AND ATRIAL FLUTTER**

| <b>RHYTHM</b> | <b>ATRIAL RATE</b> | <b>ECG CRITERIA</b>  | <b>PATIENT DEMOGRAPHICS</b>  | <b>TREATMENT</b>   |
|---------------|--------------------|--|--|--|
| MAT           | >100               | 1. $\geq 3$ p wave morphologies<br>2. Isoelectric baseline<br>3. Irregularly irregular<br>4. Irregular P-P, P-R, R-R intervals | Acute often critical illness, catecholamine excess, hypoxia, hypokalemia, hypoMg                   | Correct underlying disease.<br>Beta-blocker, magnesium, and calcium channel blockers can be considered |
| AF            | 300                | 5. "Saw tooth" flutter waves<br>6. Regular PP intervals<br>7. Regular or irregular depending on block                          | May be seen in chronic or acute cardiopulmonary disease  | Rate control   |
| Afib          | >350               | 8. Wandering baseline<br>9. Irregularly irregular RR<br>10. No discernible P waves   | May be seen in chronic or acute cardiopulmonary disease. May be seen in anatomically normal hearts | Depends on patient:<br>Rate control<br>Rhythm control<br>Cardioversion                                 |

Treatment of MAT is aimed at the underlying diseases process that caused the dysrhythmia. In contrast to Afib and AF, MAT does not usually respond to cardioversion or pharmacologic therapy. Ensuring adequate oxygenation and ventilation, repletion of potassium and magnesium, and reduction of any exogenous adrenergic stimulation should be prioritized. In addition, it is important to review the patient's medications and remove any potential medications that may precipitate MAT. As many patients with MAT have severe pulmonary disease, it is possible that some are receiving chronic therapy with a methylxanthine medication, namely, theophylline. Theophylline use and toxicity have been associated with MAT, and theophylline should be discontinued in the setting of MAT. Experts also believe that beta-agonist medications used to treat bronchospasm may propagate MAT. If possible, beta-agonist administration should be reduced if MAT develops, though this may be challenging in the patient with acute exacerbations of chronic obstructive pulmonary disease (COPD). Beta-blockers, calcium channel blockers, and magnesium sulfate have been investigated in the treatment of MAT and have shown improved ventricular rates, at the expense a reduction in blood pressure. To date, the majority of

evidence has been on the use of beta-blockers (i.e., metoprolol, esmolol) for the treatment of MAT. However, the administration of beta-blockers remains controversial in the setting of patients with COPD. Verapamil has been evaluated in the treatment of MAT. While it does reduce heart rate, it can cause a profound and sustained drop in blood pressure and should be avoided in critically ill patients. Magnesium has an acceptable adverse effect profile and has been shown to have reasonable efficacy in heart rate reduction and conversion to sinus rhythm. In general, medical therapy to reduce heart rate should be attempted after optimization and treatment of the underlying disorder.

MAT rarely causes hypotension and shock. If either is present in a critically ill patient, a search for another etiology should be performed.

## KEY POINTS

- The most common misdiagnoses for MAT are Afib and sinus tachycardia.
- MAT requires the presence of at least three morphologically different P waves in a single ECG lead.
- Treatment of MAT is aimed at the underlying disease process.
- Theophylline is associated with MAT. If possible, discontinue this medication.
- MAT rarely causes shock. If shock is present, search for another etiology.

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# DO NOT CONFUSE MOBITZ TYPE I AND MOBITZ TYPE II ATRIOVENTRICULAR BLOCK

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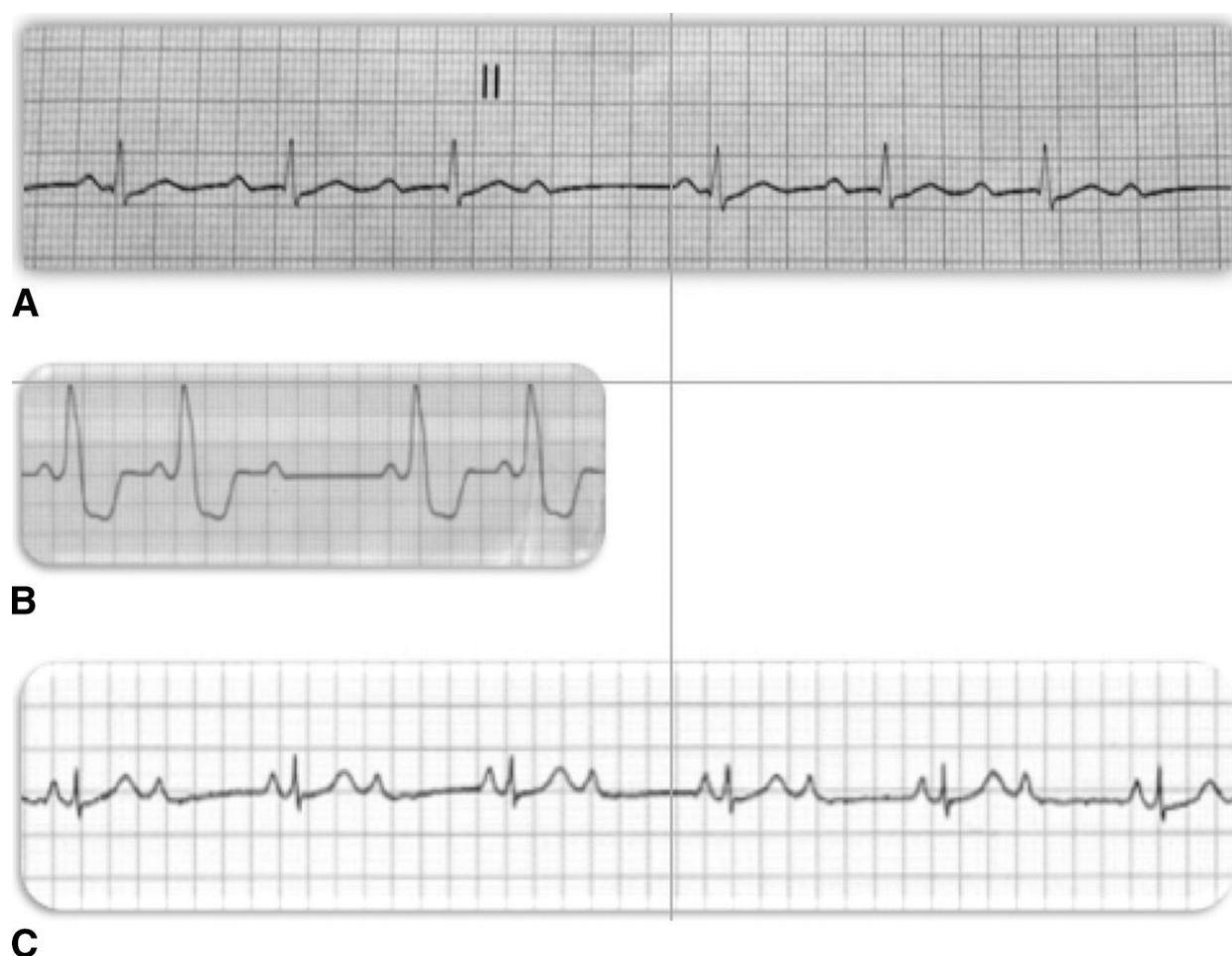
**C. BLAYKE GIBSON, MD AND J. JEREMY THOMAS, MD**

Atrioventricular block (AVB) is a broad term that encompasses several dysrhythmias that range in severity from benign to life threatening. First-degree AVB is the most common type of AVB and is characterized by a prolonged P-R interval. In the majority of patients with first-degree AVB, no treatment is necessary. In contrast to first-degree AVB, third-degree AVB is characterized by complete atrioventricular (AV) dissociation and requires immediate treatment to prevent cardiovascular collapse. Second-degree AVB is divided into two types, Mobitz type I and type II. It is important for the emergency provider to be able to distinguish between these two types of second-degree AVB, as treatment, disposition, and prognosis can be vastly different.

Mobitz type I AVB, also known as Wenckebach, is generally seen in younger patients and athletes. Though it is considered a benign dysrhythmia, it can occasionally present in older patients and be an early warning of more advanced disease of the conduction system. The electrocardiogram (ECG) characteristics of Mobitz type I AVB include the progressive lengthening of the P-R interval and the progressive shortening of the R-R interval, followed by a nonconducted P wave. This produces a characteristic and distinctive pattern of grouped beats on the ECG ([Figure 52.1A](#)). The most common location of conduction block in Mobitz type I is at the level of the AV node. Essentially, there is a gradual increase in conduction delay at the AV node, which eventually results in a nonconducted P wave and a dropped ventricular beat. Most patients with Mobitz type I AVB are asymptomatic and require no



additional evaluation or treatment. This rhythm rarely, if ever, results in hemodynamic compromise or progresses to third-degree AVB.



**Figure 52.1 A:** Mobitz type I AVB. **B:** Mobitz type II AVB. **C:** Second-degree AVB with 2:1 conduction. It is unclear if the second-degree AVB is type I or II.

Mobitz type II AVB ([Figure 52.1B](#)) is always abnormal, usually associated with significant heart disease, and has a high rate of progression to complete heart block (CHB). Mobitz type II AVB should never be considered a benign ECG finding. In contrast to Mobitz type I AVB where the conduction block is at the level of the AV node, the conduction delay of Mobitz type II is usually below the AV node. The characteristic ECG findings in type II AVB include a constant P-R interval with sporadic and unpredictable nonconducted P waves. Unlike type I AVB, there is no distinguishable ECG pattern in type II AVB. As mentioned, patients with type II AVB are at greater risk of symptomatic bradycardia and progression to CHB. For this reason, patients require hospitalization and evaluation for pacemaker placement.

*Table 52.1* lists the key ECG characteristics of Mobitz type I and type II AVB. It is important to highlight a specific scenario when there is 2:1 AVB (*Figure 52.1C*). In this case, it is difficult to determine if the AVB is type I or type II, as there is no way to assess for P-R lengthening. In this situation, it is best to treat the patient as a type II AVB until proven otherwise.

**TABLE 52.1 KEY ECG CHARACTERISTICS OF MOBITZ TYPE I AND TYPE II AVB**

|                       | <b>MOBITZ TYPE I</b>  | <b>MOBITZ TYPE II</b>                                   |
|-----------------------|---|---|
| Conduction Pattern    | Increasing P-R interval<br>Decreasing R-R interval<br>Nonconducted P wave | Unchanging P-R and R-R intervals<br>Nonconducted P wave |
| R-R Interval          | $R-R \times 2 > R\text{-Drop-R}$  | $R-R \times 2 = R\text{-Drop-R}$                        |
| Location of Block     | AV node   | Infranodal  |
| Clinical Significance | Variable, benign to concerning  | Serious   |

## KEY POINTS

- Mobitz type I AVB is characterized by a lengthening P-R interval and a shortening R-R interval followed by a nonconducted P wave.
- Mobitz type I AVB generally requires no treatment.
- Mobitz type II AVB is characterized by a constant P-R interval with unpredictable nonconducted P waves.
- Mobitz type II AVB is always abnormal and has high rate of progression to complete heart block.
- It is challenging to differentiate between type I and type II AVB when conduction is in a 2:1 pattern.

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# **BE ABLE TO RECOGNIZE ELECTROCARDIOGRAPHIC ARTIFACT FROM DYSRHYTHMIA**

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**GEORGE GLASS, MD**

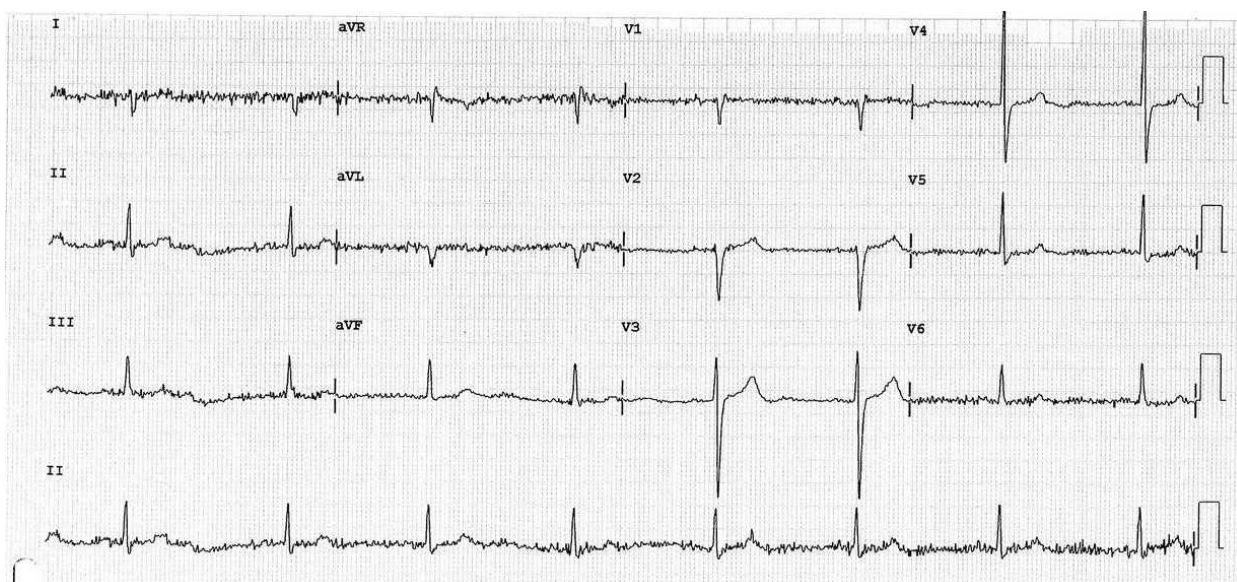
The rapid diagnosis and prompt treatment of cardiac dysrhythmias is critical to emergency medical care. Proper interpretation of rhythm strips and the electrocardiogram (ECG) can be hindered by suboptimal data collection. Rhythm strip or ECG artifact can be misdiagnosed as a dysrhythmia and result in diagnostic or therapeutic error.

ECG and rhythm strip monitors utilize skin electrodes to measure myocardial depolarization. The electrical deflections seen on these monitors can be small (~1 to 10 mV) and are prone to interference from internal and external factors. External factors that affect interpretation include equipment malfunction, interference from nearby equipment, improper lead placement, or poor electrode-skin contact due to hair, diaphoresis, or other factors such as ultrasound gel. Internal factors that affect interpretation include interference from skin or muscle due to patient movement, tremor, and shivering or from implantable devices such as a deep brain stimulator. Artifact is especially ubiquitous among critically ill patients, in whom data acquisition may occur simultaneously with other procedures such as central line placement or even chest compressions.

Accurate interpretation of the ECG or rhythm strip requires adequate data acquisition. Care should be taken to optimize this process. ECG motion artifact is often recognized and corrected immediately upon acquisition. Limiting patient movement and optimizing skin-electrode contact, through shaving and drying the skin or applying tape or other adhesive over loosened

leads, can minimize artifact during data acquisition.

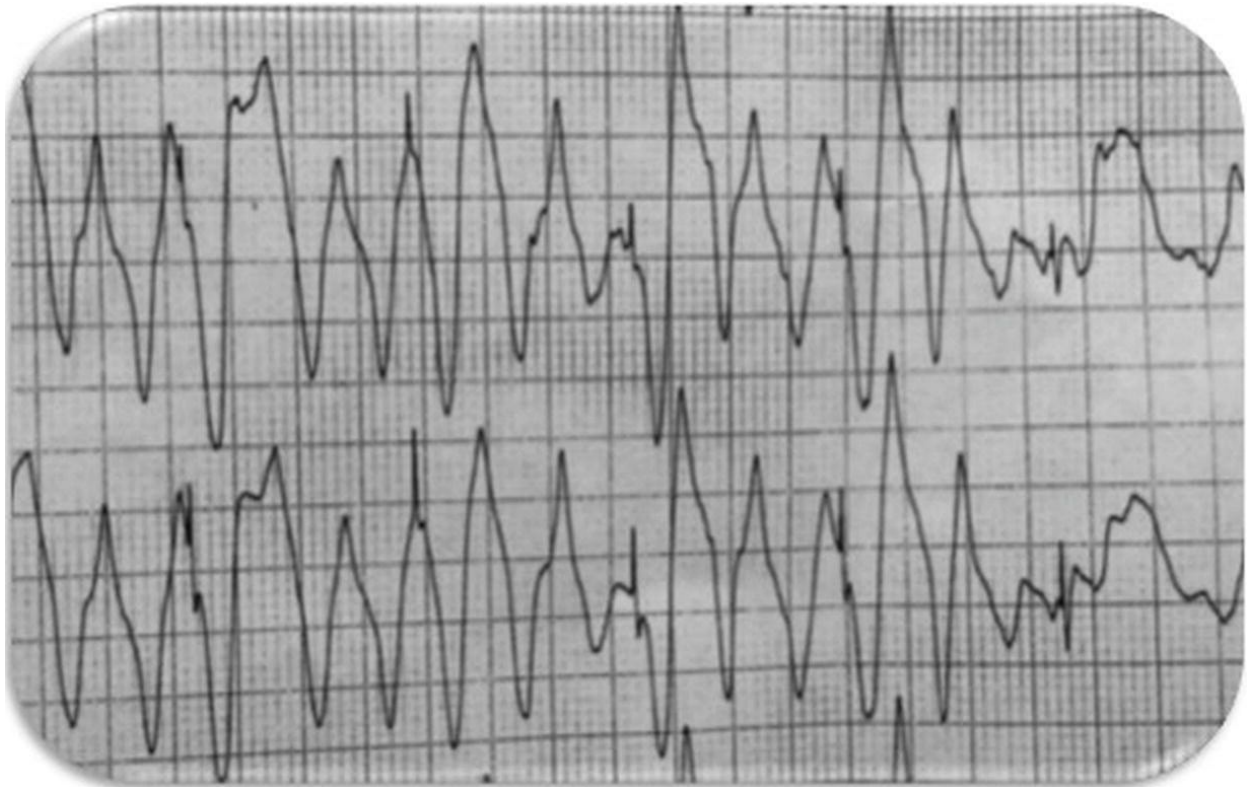
Emergency department ECGs are often presented to providers for rapid evaluation of as part of an ST-segment elevation myocardial infarction screening protocol. Care should be taken in these interpretations, as observation of the patient during ECG acquisition can provide crucial clinical information. For example, an ECG of a patient with fine tremors or shivering may have the appearance of atrial fibrillation or atrial flutter on the ECG. Observation of tremors, however, may prompt closer examination of the ECG tracing and reveal hidden P waves and a consistent R-R interval ([Figure 53.1](#)). Recognition of this “pseudofibrillation” is imperative, as an inappropriate diagnosis of atrial fibrillation may result in potentially dangerous and unnecessary therapies.



**Figure 53.1** ECG of a patient with fine resting tremor. Computerized interpretation incorrectly identifies atrial fibrillation at a rate of 130 bpm. On careful examination, P waves are evident with a regular R-R interval and sinus rate of ~50 bpm.

Larger patient movements may cause the ECG or rhythm strip to resemble ventricular fibrillation or tachycardia ([Figure 53.2](#)). Such artifact is common in patients undergoing prolonged telemetry monitoring, as they often perform activities of daily living while wearing monitor leads. In these cases, it is important to determine whether the tracing correlates with the patient’s pulse. In addition to palpation of the patient’s pulse, pulse oximetry waveforms and arterial blood pressure monitoring may be useful. A normal arterial or pulse oximetry waveform in the setting of a suspicious ECG that suggests ventricular fibrillation should raise skepticism regarding the

accuracy of the tracing. Other factors that suggest ECG artifact rather than true dysrhythmia include the presence of normal QRS complexes buried within the tracing. Furthermore, artifact should be suspected when a dysrhythmia is present in an isolated limb lead.



**Figure 53.2** Patient motion artifact resembling ventricular tachycardia. On careful examination, QRS complexes are evident within the large motion artifact–related waveforms.

## KEY POINTS

- Limit patient movement during ECG acquisition to minimize artifact.
- Optimize skin-electrode contact to minimize artifact. Consider shaving the chest or applying tape over the leads to ensure consistent contact with the skin.
- A normal pulse oximetry waveform can be helpful in assessing for ECG artifact.
- The presence of QRS complexes occurring at regular R-R intervals within a bizarre waveform suggests artifact.
- Suspect artifact when the abnormal rhythm is present in only one ECG lead.

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# **MANAGEMENT OF ATRIAL FIBRILLATION: RATE CONTROL VERSUS RHYTHM CONVERSION**

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**CHARLES KHOURY, MD, MSHA AND J. JEREMY  
THOMAS, MD**

Atrial fibrillation (AF) is the most common dysrhythmia encountered in emergency medicine. Patients with AF can present to the emergency department (ED) with a myriad of signs and symptoms that range from asymptomatic to hemodynamic collapse. In addition, patients with AF who are inadequately anticoagulated are at significant risk for catastrophic thromboembolic events. For these reasons, it is imperative that the emergency provider is knowledgeable regarding the management of patients with AF.

The ED management of patients with AF often centers on rate control or rhythm control. Both treatment strategies have been shown to improve symptoms and reduce thromboembolic events, though neither strategy has been proven to improve mortality. In addition, there is no difference between rate control and rhythm control in the occurrence of embolic events. Rate control involves the use of pharmacologic agents to decrease conduction at the atrioventricular node, thereby resulting in a lower ventricular rate. Beta-blockers and calcium channel blockers are the most common medications used to achieve rate control. Digoxin and amiodarone are also used in select patients with AF to control the heart rate. Rhythm control may be more challenging in the acute setting and involves the administration of pharmacologic agents or electrical cardioversion to restore sinus rhythm. Before attempting rhythm control, it is critical to ascertain the duration of

AF. Patients with AF for longer than 48 hours are at increased risk of thromboembolic events. For this reason, patients with AF for longer than 48 hours should be adequately anticoagulated prior to rhythm control. In addition, patients who receive rhythm control treatment often require medications to control rate in the event they revert to AF.

Electrical cardioversion is the treatment of choice for patients who are hemodynamically unstable due to AF. For the remaining patients, the decision to use rate control or rhythm control can be challenging. It is important to consider the patient's age, comorbid conditions, time of onset of AF, and anticoagulation status. For healthy patients without significant comorbid conditions who present with AF <48 hours in duration, rhythm control is the preferred approach. Both pharmacologic agents and electrical cardioversion are effective in restoring sinus rhythm. One pharmacologic approach for rhythm control that has been shown to be safe and effective is the administration of 1 g of procainamide intravenously over 60 minutes. For young patients with AF <48 hours who are treated successfully with rhythm control, long-term anticoagulation is generally not required.

For the majority of patients who present to the ED with AF of unknown duration, rate control is preferred. The intravenous administration of a beta-blocker (i.e., metoprolol, esmolol) or calcium channel blocker (i.e., diltiazem) is used to lower the ventricular rate. Recent literature has demonstrated that diltiazem may be superior to metoprolol in achieving rate control for ED patients with AF. Both classes of medications can produce hypotension. When using diltiazem, the administration of calcium prior to diltiazem has been shown to reduce the incidence of hypotension in AF patients. Digoxin can be used for patients who cannot tolerate or have contraindications to a beta-blocker or calcium channel blocker.

Regardless of the method used to treat AF, it is important to assess the patient's need for long-term anticoagulation to prevent thromboembolic events. A patient's risk for stroke can be calculated using a CHADS<sub>2</sub> or a CHA<sub>2</sub>DS<sub>2</sub>-VASc score. These calculations should be used to assess a patient's need for anticoagulation therapy to prevent embolic events, namely, stroke.

## KEY POINTS

- Electrical cardioversion is the treatment of choice for patients who are hemodynamically unstable secondary to AF.
- Consider rhythm control for healthy patients who present within 48

hours of the onset of AF.

- Diltiazem may be superior to metoprolol for rate control of patients with AF and a rapid ventricular rate.
- Consider the administration of calcium prior to diltiazem to decrease the incidence of hypotension in patients with AF.
- Use the CHADS<sub>2</sub> or CHA<sub>2</sub>DS<sub>2</sub>-VASc score to determine the stroke risk for patients with AF.

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# MANAGEMENT OF ATRIAL FIBRILLATION WITH RAPID VENTRICULAR RESPONSE

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**BRIAN L. BAUERBAND, MD AND J. JEREMY THOMAS,  
MD**

Atrial fibrillation with a rapid ventricular response (RVR) is a common condition in emergency department (ED) patients. In the initial assessment of patients with atrial fibrillation and an RVR, it is critical for the emergency provider (EP) to determine if the patient is stable or unstable. In addition, it is also important for the EP to determine if the instability is due to the RVR or is secondary to a nonarrhythmic event. Patients with atrial fibrillation who are unstable due to an RVR should be treated with synchronized cardioversion. Patients with atrial fibrillation who are unstable due to a nonarrhythmic process (i.e., sepsis, hypovolemia, alcohol withdrawal, pulmonary embolism) should receive treatment targeted to the primary event. In some of these patients, cardioversion may still be necessary while treating the primary disorder. For stable patients with atrial fibrillation and an RVR, the EP must decide upon appropriate pharmacologic therapy to control the heart rate.

Selection of the appropriate medication to control heart rate requires careful consideration of the clinical presentation, patient comorbid conditions, and current medications. Comorbid conditions such as congestive heart failure (CHF) impact the selection of medications used for rate control. Medications commonly used to control heart rate in atrial fibrillation include calcium channel blockers (CCBs), beta-adrenergic blockers, digoxin, and amiodarone.



## **CALCIUM CHANNEL BLOCKERS**

A nondihydropyridine CCBs, such as diltiazem or verapamil, is considered first-line therapy for rate control of atrial fibrillation. CCBs target the atrioventricular (AV) node and suppress cardiac conduction. Diltiazem is the recommended CCB for atrial fibrillation with an RVR. The initial dose of diltiazem is 0.25 mg/kg administered intravenously over 2 to 5 minutes. A decrease in heart rate should be seen in ~5 to 10 minutes. If there is no response, a second dose of diltiazem at 0.35 mg/kg may be given 15 to 30 minutes after the initial dose. Diltiazem can be given as a continuous infusion. The infusion is started at 5 mg/hour and can be gradually increased to 15 mg/hour based on the clinical response. Verapamil can also be used to control heart rate in atrial fibrillation. The dose of verapamil is 0.075 to 0.15 mg/kg administered intravenously over 2 to 5 minutes. The onset of action of verapamil is generally within 2 minutes of intravenous administration. Additional doses can be given every 15 to 30 minutes, as needed. A continuous infusion of verapamil can be initiated at 0.005 mg/kg/minute.

Importantly, CCBs are contraindicated for the treatment of atrial fibrillation in the setting of severe left ventricular dysfunction and in patients with Wolff-Parkinson-White (WPW) syndrome, where conduction is through the accessory pathway.

## **BETA-ADRENERGIC BLOCKERS**

Beta-adrenergic blockers can also be used to control heart rate in patients with atrial fibrillation and an RVR. Beta-adrenergic blockers control heart rate through a decrease in sympathetic tone. The most common beta-blockers used in the ED are metoprolol, propranolol, and esmolol.

Metoprolol is often the initial beta-blocker used in the management of the atrial fibrillation patient with RVR. The initial dose of metoprolol is 2.5 to 5 mg administered intravenously over ~2 minutes. Additional doses can be given every 5 minutes until a cumulative dose of 15 mg is reached. A dose of 25 mg of immediate release, or 50 mg of extended release, metoprolol can be administered as a transition to oral therapy once heart rate control is achieved. If propranolol is chosen, it can be given as an initial dose of 1 mg intravenously over 1 minute, with repeated doses of 1 mg every 2 minutes until a total of 3 mg has been given. Additional doses of propranolol can be given but should not be administered for ~4 hours from the last dose. Esmolol has the shorted duration of effect and, therefore, is of significant value when beta-blocker-related hemodynamic tolerance is a concern. Esmolol can be administered as an initial bolus of 0.5 mg/kg infused over 1

minute, followed by a continuous infusion of 50 mcg/kg/minute. The maintenance infusion of esmolol typically ranges between 50 mcg/kg/minute and 300 mcg/kg/minute.

There is no significant literature that demonstrates superiority of any one beta-adrenergic blocker. Contraindications to beta-adrenergic blocker use include hypotension, severe CHF, pulmonary edema, and severe obstructive lung disease.

## **DIGOXIN**

Digoxin is considered a second-line agent for rate control in the patient with atrial fibrillation and RVR. Digoxin should be considered in patients unable to tolerate beta-adrenergic blockers or CCBs, such as those with acute CHF exacerbations or hypotension. Digoxin lacks the efficacy of first-line agents due to a slower onset of action and a weaker potency to block the AV node.

The initial dose of digoxin is 0.25 mg administered intravenously every 2 hours to a maximum of 1.5 mg in 24 hours. The time of onset of digoxin ranges between 15 and 30 minutes, though significant rate control may not be seen for 6 to 12 hours.

## **AMIODARONE**

Amiodarone has been used to maintain sinus rhythm in patients with atrial fibrillation. It can also be used to control heart rate in atrial fibrillation patients with an RVR. Similar to digoxin, amiodarone can be considered in hypotensive patients and those with acute decompensation of CHF. The initial dose of amiodarone for this indication is 150 mg administered intravenously over 10 to 20 minutes. This dose can then be followed by an additional intravenous dose of 150 mg if adequate rate control has not been achieved within 60 minutes. A maintenance infusion of amiodarone can be started at 1 mg/minute for 6 hours, followed by 0.5 mg/minute for an additional 18 hours.

## **WOLFF-PARKINSON-WHITE PATIENTS**

WPW with accessory pathway conduction must be considered in atrial fibrillation patients with an RVR where the QRS is wide with varied morphologies. In this special situation, the use of all AV node–blocking agents should be avoided and the patient treated with synchronized cardioversion of intravenous procainamide.

## KEY POINTS

- Patients with atrial fibrillation who are unstable due to an RVR should be treated with synchronized cardioversion.
- Diltiazem, at a dose of 0.25 mg/kg, is the first-line medication for most patients with atrial fibrillation with an RVR.
- All AV node–blocking medications are contraindicated for rate control of atrial fibrillation in the setting of WPW, where conduction occurs through the accessory pathway.
- Contraindications to the use of beta-blockers in the treatment of atrial fibrillation include hypotension, severe CHF, severe COPD, and acute asthma exacerbation.
- Consider digoxin for rate control in the patient with hypotension or an acute exacerbation of CHF.

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# ATRIAL FIBRILLATION IN THE WOLFF-PARKINSON-WHITE SYNDROME

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**WILLIAM J. BRADY, MD, FACEP, FAAEM AND  
HEATHER T. STREICH, MD**

In 1930, Wolff, Parkinson, and White described the combination of bundle-branch block, shortened PR interval, and recurrent episodes of tachycardia that occurred in young, healthy patients with structurally normal hearts. This combination of electrocardiographic (ECG) findings described the ventricular pre-excitation syndrome known as the Wolff-Parkinson-White (WPW) syndrome. In WPW, an accessory pathway connects the atrial tissue to the ventricular myocardium. This connection bypasses the atrioventricular (AV) node and creates a direct electrical connection between the atria and ventricles. Patients with WPW can experience a range of tachydysrhythmias, which can lead to disabling symptoms, cardiovascular collapse, and death in select cases. The three hallmark ECG features of WPW include

- 1) PR interval  $<0.12$  seconds
- 2) Delta wave (initial slurring of the QRS complex)
- 3) Wide QRS complex (width  $>0.10$  seconds)

The PR interval is short because the impulse that progresses down the accessory pathway arrives earlier than anticipated to the ventricle. This portion of the impulse is not subjected to the physiologic slowing that occurs in the AV node. The delta wave represents activation of a portion of the ventricle through the accessory pathway. Simultaneously, there is the impulse that travels through the normal pathway into the AV node and

innervates the remainder of the ventricle. This is represented by the middle and terminal segments of the QRS complex. Ultimately, the ventricle is activated by two distinct pathways and results in a QRS complex that is a fusion of the two impulses. Importantly, only a portion of patients with the classic ECG triad of WPW are diagnosed with the syndrome. Patients must have symptoms consistent with a tachydysrhythmia (i.e., palpitations, syncope) to be diagnosed with WPW syndrome.

The most common tachydysrhythmias seen in WPW patients include a narrow complex atrioventricular reciprocating tachycardia (AVRT), atrial fibrillation, and a wide-complex AVRT. Atrial fibrillation can be seen in up to 25% of patients with WPW syndrome. In atrial fibrillation, the multiple foci of atrial impulses can be transmitted through the accessory pathway and result in an uncontrolled ventricular rate. When these uncontrolled ventricular depolarizations occur along with those that arrive via the AV node, patients can experience rapid ventricular rates and cardiovascular collapse. In patients with WPW syndrome who experience atrial fibrillation, the ECG demonstrates several unique features that include

- 1) Very rapid, irregularly irregular rhythm
- 2) Wide QRS complex
- 3) Significant beat-to-beat variation in QRS complex morphologies

A delta wave can also be seen in these patients.

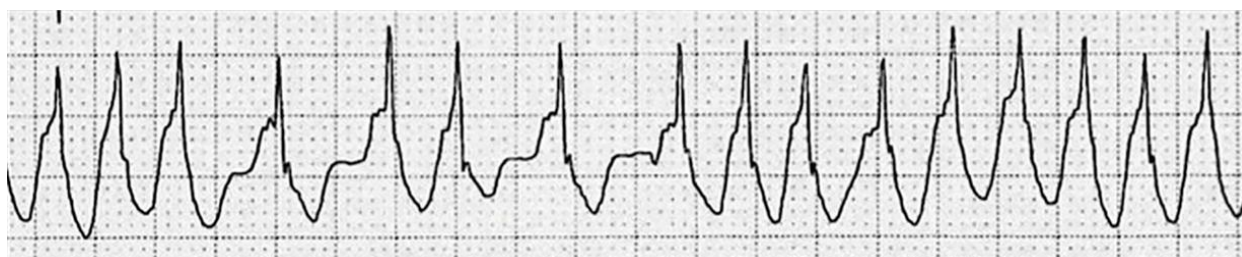
The management of WPW-related atrial fibrillation is largely based on the hemodynamic status of the patient. In the hemodynamically unstable patient, cardioversion with sedation is the treatment of choice. In the stable patient, rate control with medications can be attempted, though cardioversion and other resuscitation interventions should be immediately available. Procainamide is the primary agent for control of WPW-related atrial fibrillation in the stable patient. Procainamide is dosed at 20 to 30 mg/minute, until the dysrhythmia is terminated or one of the following end points is reached: development of hypotension, the QRS complex widens by 50% or more from its original width, acceleration of the tachycardia, or a total of 1 g is administered. Patients who are treated with procainamide should be placed on continuous cardiac monitor and have frequent assessments of blood pressure. Regardless of the administration strategy, procainamide has a relatively slow onset of action and may not reach therapeutic blood levels for 40 to 60 minutes.

Amiodarone should be avoided in the treatment of patients with WPW-related atrial fibrillation. Amiodarone was previously recommended as an appropriate agent in this setting. However, its diverse electrophysiologic

effects (i.e., beta-adrenergic, calcium channel, fast sodium channel blocking mechanisms) impact the accessory pathway and make rapid intravenous administration dangerous. Amiodarone can accelerate the ventricular rate and result in ventricular fibrillation or cardiovascular collapse.

Medications that block the AV node are contraindicated in the treatment of patients with WPW and atrial fibrillation. Calcium channel antagonists, beta-adrenergic blocking agents, adenosine, and digoxin can enhance conduction via the accessory pathway, and lead to rapid ventricular rates, malignant ventricular dysrhythmias, and cardiovascular collapse. Once the patient is stabilized and the rhythm converted to sinus rhythm, admission to a monitored critical care bed is most appropriate. Cardiology consultation should also be obtained.

Atrial fibrillation occurring in the setting of the WPW syndrome should be included in the differential diagnosis of patients with a wide-complex tachycardia. Clinical clues to the diagnosis include a young patient who presents with a very rapid, irregularly irregular rhythm that has significant beat-to-beat variation in the QRS complex morphology ([Figure 56.1](#)).



**Figure 56.1** Atrial fibrillation in the WPW syndrome is characterized by a very rapid, irregularly irregular rhythm with a widened QRS complex and beat-to-beat variation in the QRS complex morphology.

## KEY POINTS

- The classic ECG triad of WPW includes a short PR interval, delta wave, and a widened QRS complex.
- Atrial fibrillation can occur in up to 25% of patients with WPW syndrome.
- Cardioversion is the treatment of choice for unstable patients with WPW-related atrial fibrillation.
- Procainamide is the primary medication for control of WPW-related atrial fibrillation in the stable patient.
- All medications that block the AV node should be avoided in the

treatment of patients with WPW-related atrial fibrillation.

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# NEVER MISTAKE VENTRICULAR TACHYCARDIA FOR SUPRAVENTRICULAR TACHYCARDIA WITH ABERRANT CONDUCTION

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**HEATHER GROTH, MD**

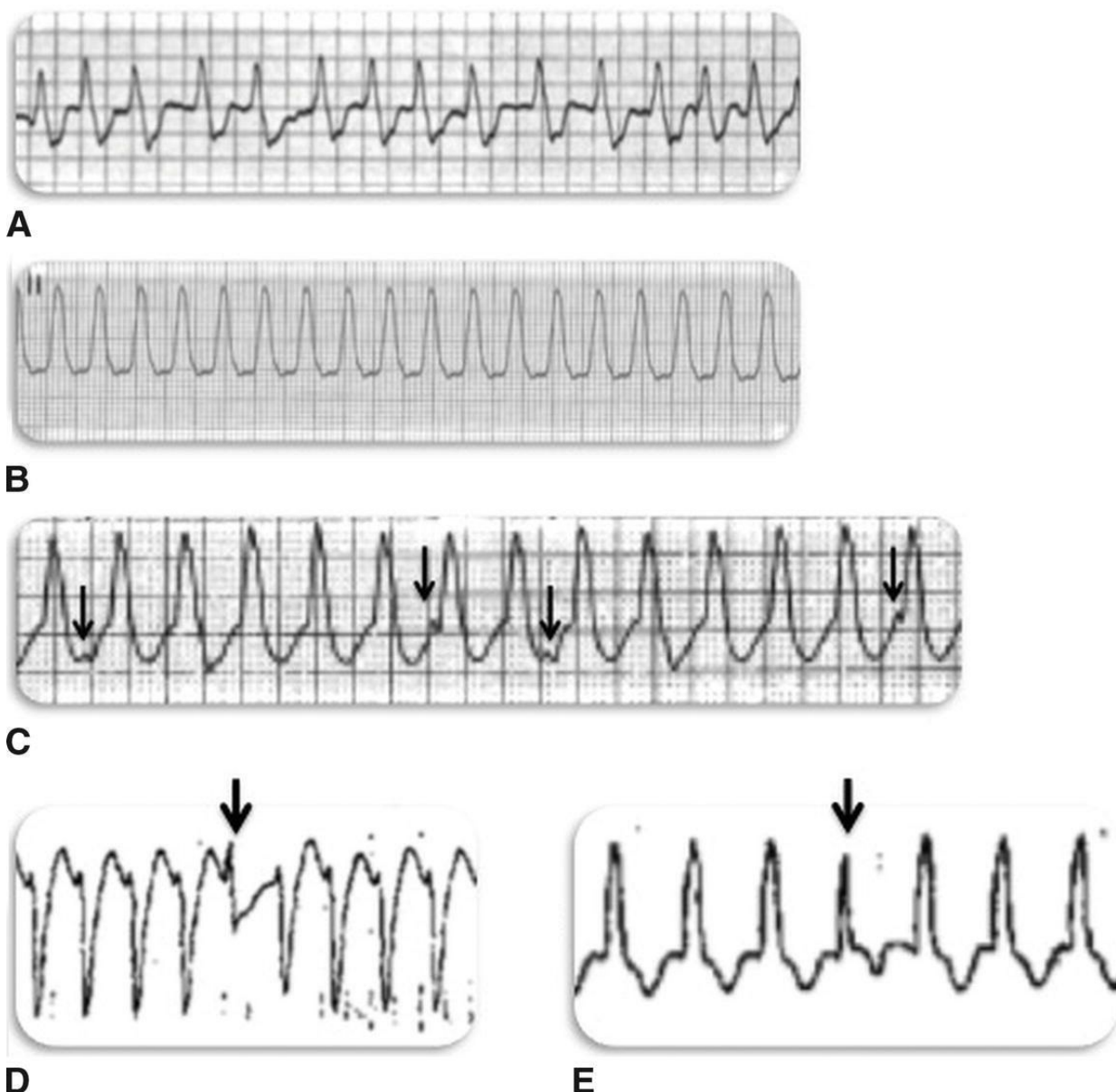
Evaluation of the emergency department patient with a wide-complex tachycardia (WCT) can be a challenge for any emergency provider (EP). It can be especially difficult to distinguish between ventricular tachycardia (VT) and supraventricular tachycardia (SVT) with aberrant conduction. Fortunately, if the patient is unstable, the initial treatment for both rhythms is synchronized electrical cardioversion. However, for the stable patient with a WCT, misdiagnosis of VT as SVT with aberrant conduction could prove to be a deadly error.

A WCT is defined as a ventricular rate >100 beats per minute and a QRS complex duration of more than 0.12 seconds. The primary differential for a WCT is VT and SVT with aberrant conduction. Rhythms that can produce SVT with aberrant conduction include paroxysmal SVT, atrial fibrillation, atrial flutter, sinus tachycardia, and hyperkalemia. Though it may often seem impossible to differentiate VT from SVT with aberrant conduction, there are a number of electrocardiography (ECG) and historical features that may be used to improve the EP's ability to differentiate between these two rhythms.

Importantly, there is no single data point from the history, physical examination, or ECG that can reliably distinguish VT from SVT with aberrant conduction. Notwithstanding, age >50 years and a history of prior myocardial infarction, ischemic heart disease, structural heart disease, or VT



all increase the likelihood of VT. Aside from age and components of the past medical history, ECG findings are generally used to help differentiate VT from SVT with aberrant conduction. Multiple ECG abnormalities have been proposed to differentiate these rhythms. The regularity of the rhythm can be helpful. An irregularly irregular rhythm can suggest SVT with aberrant conduction due to atrial fibrillation ([Figure 57.1A](#)). In contrast, monomorphic VT is generally a regular rhythm ([Figure 57.1B](#)). Additional findings that suggest VT include QRS concordance in the precordial leads, atrioventricular (AV) dissociation ([Figure 57.1C](#)), capture beats ([Figure 57.1D](#)), and fusion beats ([Figure 57.1E](#)). These findings, though poorly sensitive, are highly specific for VT. Brugada and colleagues developed one of the most frequently cited decision rules to distinguish between VT and SVT with aberrant production. In the Brugada algorithm, VT is diagnosed if the ECG demonstrates any of the following four criteria:



**Figure 57.1** Various ECG features that can be used in the differentiation of VT from SVT with aberrant conduction. **A:** An irregularly irregular rhythm with widened QRS complex, consistent with atrial fibrillation with bundle-branch block. **B:** Monomorphic VT. **C:** AV dissociation; *arrows* denote the P waves. **D:** Capture beat (*arrow*). **E:** Fusion beat (*arrow*).

- 1) Is there absence of an RS complex in leads V1 through V6?
- 2) Is there an R to S interval >100 ms in one precordial lead?
- 3) Is there AV dissociation?
- 4) Are there morphology criteria for VT?

In practice, the Brugada Criteria can be difficult to recall, and validation studies have shown significantly lower sensitivity and specificity than

originally published by Brugada and colleagues. In fact, a recent study showed 22% disagreement among EPs who used the Brugada Criteria in the evaluation of WCT.

Additional algorithms have been published, including those by Verecke and colleagues and by Griffith and colleagues. These algorithms can be easily found online. In the Griffith algorithm, SVT with aberrant conduction is diagnosed only if the QRS axis and morphology meet criteria for a right bundle-branch block or left bundle-branch block and there is no evidence of AV dissociation. Otherwise, all rhythms are considered VT. Importantly, no ECG criteria or algorithms have been shown to be 100% specific for SVT or VT.

Hemodynamic stability should also not be used to differentiate VT from SVT with aberrant conduction. Hemodynamic stability is not universally associated with SVT with aberrant conduction and is not indicative of rhythm classification. In addition, medications used to treat SVT (i.e., adenosine, calcium channel blockers) could cause decompensation if given to a patient with VT. Conversely, medications generally used to treat VT (i.e., procainamide, amiodarone) can be effective in the treatment of a patient with SVT with aberrant conduction. When there is clinical uncertainty, a regular WCT should be treated as VT until proven otherwise.

## KEY POINTS

- A regular WCT should be assumed to be VT until proven otherwise.
- There is no single feature of the history, physical examination, or ECG that can reliably distinguish VT from SVT with aberrant conduction.
- Age >50 years and a history of prior myocardial infarction, ischemic heart disease, structural heart disease, or VT increases the likelihood of VT.
- There are currently no published ECG algorithms that routinely distinguish VT from SVT with aberrant conduction.
- Hemodynamic stability should not be used to differentiate VT from SVT with aberrant conduction.

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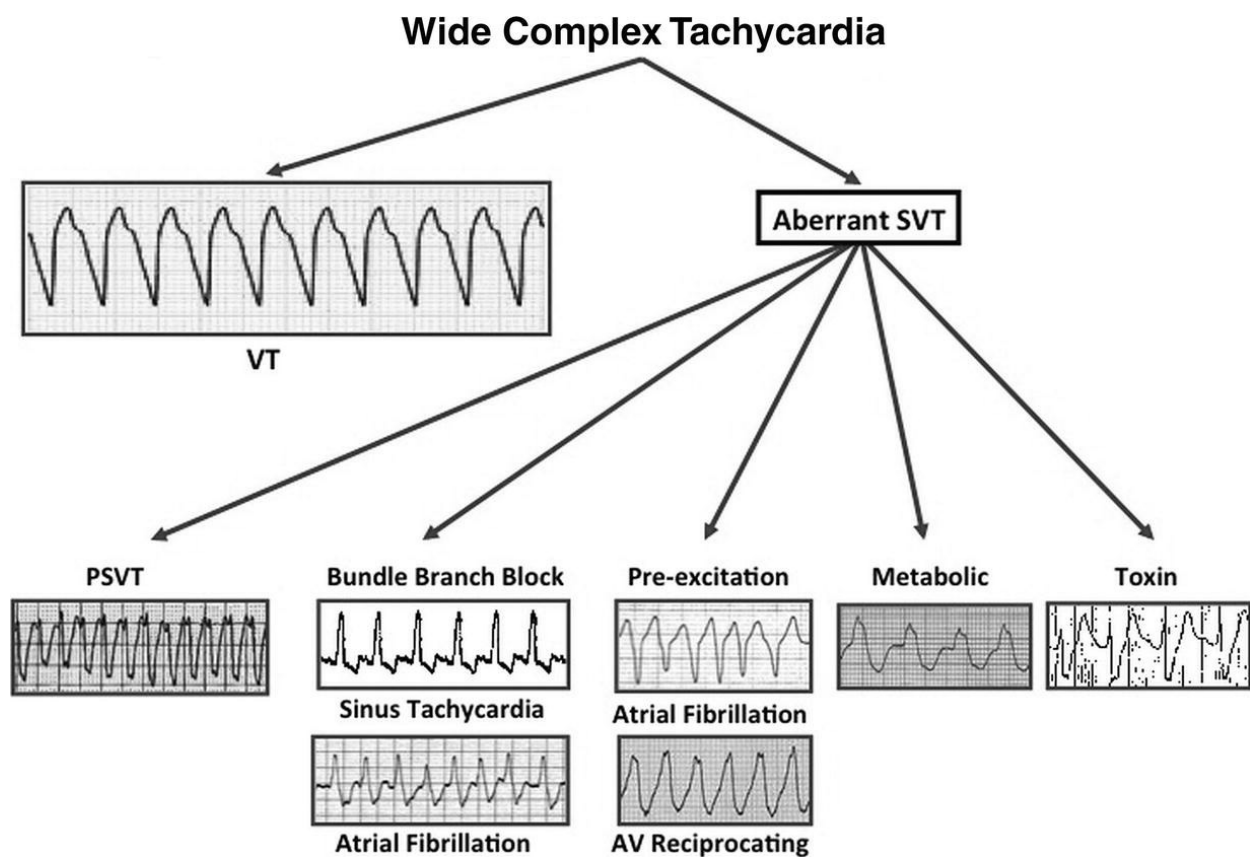
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# **KNOW THE MIMICS OF VENTRICULAR TACHYCARDIA**

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Ventricular tachycardia is a wide complex tachycardia (WCT) that is typically associated with coronary artery disease or other significant heart disease. The rapid rate, often in conjunction with poor baseline cardiac function, can produce cardiovascular instability. If this instability is not promptly treated, cardiovascular collapse will almost certainly ensue. This has led to the position of treating all WCTs as ventricular tachycardia (VT) until proven otherwise. For management purposes, it is important to attempt to differentiate between VT and supraventricular tachycardia (SVT) with aberrant conduction. In some cases, the distinction between these two rhythms is not possible. As a result, management decisions must be made based upon the patient's clinical situation and the electrocardiography (ECG) rhythm. The differential diagnosis of WCT is depicted in [Figure 58.1](#).



**Figure 58.1** The differential diagnosis of WCT with ventricular tachycardia and supraventricular tachycardia with aberrant conduction. The dysrhythmias associated with supraventricular tachycardia with aberrant conduction include paroxysmal supraventricular tachycardia, sinus tachycardia, atrial fibrillation with rapid ventricular response, Wolf-Parkinson-White syndrome WCTs, and metabolic and toxicologic dysrhythmias.

There are numerous conditions that can mimic the appearance of VT on the ECG. These include, but are not limited to, SVT with aberrant conduction, Wolff-Parkinson-White syndrome, artifact, metabolic disorders (i.e., hyperkalemia), and toxic ingestions (i.e., medications that block the sodium channel). In some of these conditions, the electrical impulse is delayed, or slowed, as it passes through the conduction system and ventricular myocardium. This results in a wide QRS complex. The dysfunction in the intraventricular conduction system can be permanent or temporary. In some cases, abnormal conduction may only be seen at higher heart rates.

SVTs with aberrant conduction can be regular or irregular and include sinus tachycardia, atrial fibrillation, paroxysmal SVT, atrial flutter, and multifocal atrial tachycardia. Due to the presence of a bundle branch block (BBB), SVTs due to these rhythms will demonstrate a wide QRS complex.

Wolff-Parkinson-White syndrome (WPW) is a form of ventricular pre-excitation that involves an accessory conduction pathway between the atria and ventricles. WPW patients are prone to develop a variety of supraventricular tachyarrhythmias, especially WPW-related atrial fibrillation and WCT. Severe hyperkalemia can also cause a rapid, wide complex rhythm that can easily be mistaken for VT. Hyperkalemia should be considered in any WCT where the heart rate is <120 beats per minute (bpm). Medications that block the sodium channel (i.e., tricyclic antidepressants) produce a WCT that is often mistaken for VT. In the case of hyperkalemia or sodium channel blocker toxicity, the administration of sodium bicarbonate can be both diagnostic and therapeutic. Sinus tachycardia with an anterior ST-segment elevation myocardial infarction can mimic VT. Lastly, ECG artifact can be mistaken for VT. Patient movement, poor electrode application, equipment malfunction, or electromagnetic interference can all cause ECG artifact and mimic the appearance of VT.

## KEY POINTS

- SVT with aberrant conduction is a common mimic of VT.
- Consider hyperkalemia in any WCT where the heart rate is <120 bpm.
- Tricyclic antidepressant toxicity is well known to cause a WCT and should be considered in the differential diagnosis of VT.
- Patient movement, equipment malfunction, and poor electrode placement or contact cause result in artifact and mimic the appearance of VT.
- Consider WPW in the differential diagnosis of a WCT.

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# **DO NOT EXCLUDE CARDIAC CAUSES OF CHEST PAIN BECAUSE THE PATIENT DOES NOT HAVE TRADITIONAL RISK FACTORS FOR ACUTE CORONARY SYNDROME**

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Traditional risk factors for coronary artery disease (CAD) have been described by the Framingham Heart Study and include age, gender, hyperlipidemia, hypertension, diabetes, smoking, and family history of heart disease. These risk factors have been incorporated into clinical guidelines, as well as the daily practice of the emergency provider (EP) to quantify the risk for acute coronary syndrome (ACS) in the emergency department (ED) patient with acute chest pain. When objective evidence clearly demonstrates an ACS, there is little uncertainty as to how the EP should proceed with treatment and disposition. However, the absence of typical ischemic electrocardiography (ECG) findings along with negative results of cardiac biomarker tests does not exclude ACS. This scenario often presents a diagnostic challenge to even the most astute clinician. The combination of clinical features, CAD risk factors, and clinical decision rules (i.e., PURSUIT, TIMI, GRACE, FRISC, HEART Score) is often used by the EP to risk stratify patients and determine an appropriate patient disposition. The HEART score was the first risk stratification tool derived from an ED population, emphasized clinician gestalt, incorporated non-ACS chest pain,



and evaluated patient-centered outcomes that are important to the EP. However, it is a tool that continues to utilize traditional risk factor elements that have not been shown to be helpful in the assessment of ED patients with suspected ACS.

In the ED patient with acute chest pain, the EP develops a clinical gestalt for ACS based upon individual elements from the history, physical examination, and ECG. In regard to traditional risk factors for ACS, it is important to note that these were derived from several longitudinal studies that predict the incidence of CAD over the course of many years, even decades. These risk factors were not evaluated in the diagnosis of ACS in the patient with acute chest pain. There remains limited emergency medicine literature as to whether the presence of risk factors for ACS results in over- or underdiagnosis, along with an increase in potentially avoidable admissions.

When compared to ischemic ECG abnormalities, risk factors for CAD provide limited information and predictive value in the ED patient with suspected ACS. In fact, no individual risk factor has been shown to significantly increase the likelihood of ACS in women. For men, Jayes and colleagues reported that only a history of diabetes and a family history for myocardial infarction (MI) significantly increased the relative risk for ACS. In the same study, a history of hypercholesterolemia, cigarette smoking, hypertension, and a family history of MI in those younger than 50 years of age did not significantly alter the relative risk for MI.

In a recent multivariate analysis, Body and colleagues found no increased incidence of acute MI in patients with an increasing number of CAD risk factors. Additionally, the absence of any risk factor for CAD only had a negative likelihood ratio of 0.61. Similarly, Han and colleagues demonstrated that the total number of patient risk factors for CAD had poor diagnostic performance, except in those younger than 40 years of age. For those older than 65 years of age, the number of risk factors was less useful. Most importantly, ACS cannot be excluded simply based on the absence of traditional risk factors for CAD.

It is imperative to obtain a complete medical history in ED patients with acute chest pain. Notwithstanding, the presence or absence of traditional risk factors for ACS fails to significantly augment clinical gestalt based on the history of present illness and initial ECG. Simply put, the absence of CAD risk factors does not predict a low risk for ACS, even in young patients. Future research and clinical decision tools, such as the Manchester Acute Coronary Syndromes (MACS), should focus on objective clinical and diagnostic components that are more predictive of ACS.

## KEY POINTS

- Traditional CAD risk factors are not predictive of ACS in the ED patient with acute chest pain.
- A lack of CAD risk factors should not be used to exclude the diagnosis of ACS in ED patients with acute chest pain.
- An increasing number of risk factors does not predict a higher likelihood of ACS in the ED patient with acute chest pain.
- Current clinical decision rules still incorporate traditional risk factors to predict major adverse cardiac events over the next 6 weeks.
- The MACS clinical decision rule does not utilize traditional CAD risk factors and may perform better in patients with suspected ACS. This tool requires prospective validation.

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# **DO NOT FORGET TO CONSIDER NONTRADITIONAL RISK FACTORS FOR CORONARY ARTERY DISEASE IN PATIENTS WITH CHEST PAIN**

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**THOMAS HARTKA, MD, MS**

Emergency providers (EP) frequently evaluate patients with acute chest pain. While most EPs rely on traditional risk factors for coronary artery disease (CAD) to aid their evaluation, they should also be cognizant that there are nontraditional risk factors that can impact the evaluation of patients with acute chest pain. Failure to consider these nontraditional risk factors may lead EPs to inappropriately determine that a patient is at low risk for an acute coronary syndrome (ACS). These nontraditional risk factors include chronic kidney disease (CKD), radiation therapy (RT), systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), corticosteroid therapy, and human immunodeficiency virus (HIV).

## **CHRONIC KIDNEY DISEASE**

Cardiovascular disease is the leading cause of death among patients with CKD. CKD has been shown to be an independent risk factor for CAD. There is a 40% to 50% rate of significant CAD on angiogram of patients with recent-onset end-stage renal disease (ESRD). Even in mild renal impairment, there is an increased risk of CAD and ACS. Patients with Stage IV CKD have a staggering 5-year mortality of 45.7%. In fact, even in patients with Stage II CKD, the 5-year mortality is 19.5%. Patients with any degree of renal dysfunction should be considered to have an increased risk of CAD.

## **RADIATION THERAPY**

RT to the chest has been shown to be a risk factor for the development of CAD. Chest RT is a common adjunct to chemotherapy in select lymphomas and breast cancer. Risk factors for the development of radiation-induced CAD include higher dose of radiation, younger age at time of exposure, and traditional cardiac risk factors. A meta-analysis of patients from early trials of adjunct RT for breast cancer demonstrated a 27% increase in mortality from heart disease. It should be noted that radiation techniques have changed substantially since these early studies and modern RT probably confer less risk. In most cases, CAD does not develop until more than a decade after exposure. Many patients may forget to report their distal radiation treatment. It is important for EPs to inquire about RT to the chest, especially in patients who have a history of breast cancer or lymphoma.

## **SYSTEMIC LUPUS ERYTHEMATOSUS**

SLE is a common disorder that involves chronic inflammation in multiple organ systems, which often includes the cardiovascular system. It is well established that patients with SLE have an increased risk for myocardial infarction (MI). This risk is especially elevated in young female patients with SLE. A study using age-matched controls demonstrated a 50-fold increase in the rate of MI in female SLE patients aged 35 to 44 years. From autopsy studies, it is known that patients with SLE have an increased rate of atherosclerosis compared to the general population. The common treatment for SLE, chronic corticosteroid use, has also been associated with higher rates of MI in this population. EPs should have a heightened suspicion for ACS in patients with SLE, especially younger female patients.

## **RHEUMATOID ARTHRITIS**

Inflammatory joint diseases (IJD), especially RA, are associated with a substantially increased risk of CAD. Patients with IJD have a higher risk of premature death compared to the general population, and most of this risk can be attributed to CAD. A Dutch population-based study found that the development of CAD in patients with RA was similar to that of patients with diabetes mellitus (DM). A large meta-analysis showed patients with RA had a relative risk of 1.48 for developing CAD compared to patients without RA. Special care should be taken when evaluating patients with RA, or other IJD, who present with acute chest pain, as these patients have an increased risk of

CAD similar to that of patients with DM.

## CORTICOSTEROID THERAPY

Long-term corticosteroid treatment is used for the management of several conditions that include organ transplantation, inflammatory bowel disease, SLE, and RA. Many of these conditions independently increase a patient's risk of developing CAD; however, corticosteroids themselves also increase this risk. Long-term corticosteroid use is associated with metabolic and hemodynamic changes that include hyperlipidemia, insulin resistance, weight gain, and central obesity. A large case-controlled cohort study found a relative risk of acute MI to be 1.42 for patients on long-term oral corticosteroids compared to nonusers. Long-term corticosteroid should be considered a risk factor for CAD due to its side effects and the underlying disease for which they have been prescribed.

## HUMAN IMMUNODEFICIENCY VIRUS

Patients infected with HIV have been shown to be at increased risk of CAD and ACS, the etiology of which is likely multifactorial. Replication of HIV appears to directly increase the risk of CAD. A study that compared continuous with intermittent antiretroviral therapy (ART) showed a relative risk of 1.5 for nonfatal cardiovascular events for patients in the intermittent ART arm. ART is associated with hyperlipidemia, decreased high-density lipoprotein, and insulin resistance. Several large cohort studies have shown an increased rate of MI, specifically treatment with a protease inhibitor (PI). However, two large cohort studies failed to show an increased rate of MI or symptomatic heart disease in patients taking ART. Despite some conflicting data, patients with HIV and AIDS are likely at a higher risk for CAD and ACS.

### KEY POINTS

- Patients with any degree of CKD should be considered to have an increased risk of CAD.
- SLE confers a 50-fold increase in the rate of MI in female patients aged 35 to 44 years.
- Patients with IJD have a risk of CAD similar to that of patients with DM.
- Long-term corticosteroid use causes hyperlipidemia, insulin

resistance, weight gain, and central obesity and is a risk factor for CAD.

- HIV patients may be at increased risk of CAD and ACS.

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## **DO NOT FORGET ABOUT THE NON-ACS CAUSES OF CHEST PAIN**

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Annually, almost 6 million patients present to the emergency department with a chief complaint of chest pain. Thankfully, the majority will not have an acute coronary syndrome (ACS) as the etiology of their symptoms. There are numerous etiologies of chest pain that range from benign to life threatening. The emergency provider (EP) should broaden the differential of acute chest pain beyond simply ACS, in order to promptly recognize and treat additional life-threatening etiologies of chest pain.

Life-threatening causes of acute chest pain include ACS, aortic dissection, pulmonary embolism (PE), tension pneumothorax, cardiac tamponade, and esophageal rupture. Delayed diagnosis of these etiologies is associated with significant increases in patient morbidity and mortality. This chapter discusses pearls and pitfalls from the history of present illness and physical examination for the non-ACS causes of chest pain.

### **THORACIC AORTIC DISSECTION**

The incidence of thoracic aortic dissection (TAD) is ~3 cases per 100,000 people per year. The incidence of TAD peaks at age 70 and is more common in males. Patients commonly have a history of hypertension. Patients with TAD who are younger than 40 years of age commonly have a history of connective tissue disease (i.e., Marfan syndrome, Ehlers-Danlos syndrome), chronically abuse cocaine, or have a history of a bicuspid aortic valve. Pregnancy, especially in the third trimester, is an established risk factor for TAD. Additional risk factors for TAD include a prior history of aortic

surgery, aortic valve disease, or a family history of aortic valve disease.

The most common presenting symptom is the abrupt onset of severe pain. In contrast to the textbook descriptions of “tearing” or “ripping” pain, patients with TAD more commonly report the abrupt onset of sharp pain that quickly reaches maximal intensity. TAD should also be considered in any patient in whom symptoms cross the diaphragm (i.e., chest and abdominal pain). Acute onset of thoracic back pain can be reported in patients with a dissection of the descending aorta.

Classic physical examination findings in the patient with TAD include a blood pressure differential between the extremities, an extremity pulse deficit, an aortic insufficiency murmur, or a focal neurologic finding. Importantly, these findings are variable. In fact, a systolic blood pressure differential has been found to be both poorly specific and sensitive for TAD. A normal physical examination should not exclude the diagnosis of TAD when the history is strongly suggestive of this etiology.

Chest x-ray (CXR) abnormalities seen in patients with TAD include a widened mediastinum and an abnormal aortic contour. These classic findings, however, are infrequently found in the majority of cases. Computed tomography (CT) angiography is commonly used to confirm, or exclude, the diagnosis of TAD. In recent years, there have been numerous studies that have attempted to validate the use of D-dimer in the evaluation of patients with TAD. At present, a negative D-dimer is insufficient to exclude TAD in low-risk patients.

## **PULMONARY EMBOLISM**

The estimated incidence of PE continues to rise. Risk factors for PE are well documented and include a history of hypercoagulability, recent surgery or prolonged immobilization, connective tissue disease, and exogenous estrogen use.

Patients with PE commonly report dyspnea, with or without exertion, and acute chest pain. The chest pain of PE is typically described as pleuritic in character. Other historical features that may guide the EP are the presence of lower extremity or calf pain, unilateral lower extremity swelling, cough, or hemoptysis.

Similar to TAD, physical examination findings are often absent in the patient with PE. Vital sign abnormalities may raise the clinician’s suspicion, but are not always present at the time of ED evaluation. Patients with PE can have tachycardia, tachypnea, or hypoxia. With a massive PE, the patient may present with hypotension and signs of shock. Fever, when present, is



typically low grade and can mislead the EP toward a diagnosis of pneumonia. Unilateral lower extremity swelling can suggest the diagnosis of DVT and raise suspicion for PE.

The diagnostic evaluation for PE requires an understanding of current clinical decision rules. The well-versed EP should be able to exclude PE in low-risk patients with a combination of clinical gestalt and the Pulmonary Embolism Rule-out Criteria. Beyond these low-risk patients, EPs must understand and correctly apply the Well's criteria or Revised Geneva Score to calculate pretest probability and guide further evaluation with either a D-dimer test or CT angiography of the chest.

## **TENSION PNEUMOTHORAX**

The incidence of tension pneumothorax varies widely and is dependent on the population studied. The EP should have a high index of suspicion for tension pneumothorax in patients with a history of trauma or recent instrumentation of the thorax, neck, or upper extremity regions. Tension pneumothorax almost always presents with the combination of acute chest pain and respiratory distress. Clinical features include unilateral or absent breath sounds, hypotension, jugular venous distension, and tracheal deviation.

Tension pneumothorax is a clinical diagnosis that requires rapid intervention to prevent cardiac arrest and death. Bedside ultrasound can be used to quickly confirm the diagnosis in patients with an equivocal exam.

## **CARDIAC TAMPONADE**

In cardiac tamponade, a pericardial effusion increases pericardial pressure, decreases right ventricular filling, and decreases cardiac output. Etiologies of pericardial effusion include malignancy, trauma, infectious diseases, pericarditis, uremia, and acute myocardial infarction. Patients with a pericardial effusion with development of tamponade often report chest pain, dyspnea, and fatigue. Diminished breath sounds or a pericardial friction rub are heard in only one-third of patients. The classic triad of hypotension, muffled heart sounds, and jugular venous distension is a late finding in patients with tamponade. The electrocardiogram in patients with a pericardial effusion can demonstrate low voltage, tachycardia, and electrical alternans.

Similar to tension pneumothorax, bedside ultrasound can be used to quickly confirm the diagnosis. The presence of an effusion with diastolic right ventricular collapse should lead to emergent pericardiocentesis.

# ESOPHAGEAL RUPTURE

Esophageal rupture is a rare diagnosis. Common precipitants of esophageal rupture include iatrogenic (i.e., esophagogastroduodenoscopy), severe emesis, trauma, caustic ingestion, and esophageal foreign body. Chest pain is most often retrosternal, is severe, and frequently radiates to the back, neck, shoulders, or abdomen. Additional historical features may include dysphagia, dyspnea, and emesis. Patients with esophageal rupture can present in shock with tachycardia, hypotension, and signs of poor perfusion. Physical exam findings can include subcutaneous emphysema in the cervical and clavicular region. Patients with an intra-abdominal rupture may present with signs of a surgical abdomen.

Although a CXR can demonstrate a pneumothorax, pneumomediastinum, or pleural effusion, the diagnosis of esophageal rupture is confirmed with CT of the chest. CT findings of esophageal rupture can include periaortic or periesophageal air, pleural effusions, or soft tissue stranding.

## KEY POINTS

- Patients with TAD more commonly report the abrupt onset of sharp pain that quickly reaches maximal intensity.
- Patients with PE may not present with tachycardia, tachypnea, or hypoxia.
- Tension pneumothorax remains a clinical diagnosis.
- The classic triad of hypotension, muffled heart sounds, and jugular venous distension is a late finding in patients with cardiac tamponade.
- The most common etiology of esophageal rupture is iatrogenic.

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# **BE CAUTIOUS DIAGNOSING “ANXIETY” OR “PANIC DISORDER” IN PATIENTS WITH CHEST PAIN AND ANXIETY**

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**ADAM E. NEVEL, MD, MBA**

Chest pain is a common complaint in emergency department (ED) patients. Patients with acute chest pain often present with concomitant symptoms such as dyspnea, diaphoresis, anxiety, nausea, or emesis. While these additional symptoms can be helpful to the emergency provider (EP), caution should be taken when relying on any one symptom. Specifically, the association of anxiety or panic with a noncardiac cause of chest pain can be a risky assumption. While some studies have shown a high prevalence of panic disorder within an ED chest pain population (as high as 20%), anxiety and panic itself are poorly specific findings.

Patients with acute life-threatening cardiac or pulmonary conditions frequently present with feelings of panic or impending doom in association with chest pain. This may even be in the setting of a stressful situation and mislead the EP to feel confident that anxiolytic therapy and reassurance is all that may be needed. These stressful situations can be the catalyst for a more serious condition, as moments of high stress and anger have been associated with an increased incidence of cardiovascular events. These events are believed to occur from the catecholamine surge that is associated with acute emotional distress. This can, in turn, lead to increased platelet aggregation and subsequent rupture of an unstable intracoronary plaque. Furthermore, multiple studies have shown a link between anxiety disorders and higher

rates of cardiac risk factors. This suggests that patients with a psychiatric history may be at higher risk for cardiac disease.

Unfortunately, misdiagnosis of a cardiopulmonary condition as anxiety is a frequent occurrence. A recent survey-style study of emergency medicine physicians found that nearly 10% of self-reported missed diagnoses consisted of acute coronary syndrome (ACS), pulmonary embolism (PE), or aortic dissection. In addition to the patient consequences of a missed or delayed diagnosis, there are significant medicolegal implications as well. Roughly 7% of all malpractice claims for misdiagnosis are due to missed myocardial infarction (MI) or PE. With that in mind, EPs should undoubtedly think twice before excluding a life-threatening etiology of chest pain based primarily on the concomitant symptoms of anxiety or panic.

A less common, but well-described, condition involving ACS and emotional distress is takotsubo cardiomyopathy, also termed “broken-heart syndrome.” Patients with takotsubo often present with chest pain in the setting of recent emotional event (i.e., loss of a loved one). Electrocardiogram (ECG) findings in patients with takotsubo can be similar to an acute anterior wall MI. These patients may even have elevations in cardiac biomarkers and echocardiogram findings that support a diagnosis of acute infarction. Cardiac catheterization, however, reveals no culprit coronary lesions. Instead, dilatation of the left ventricle is seen with a pathognomonic appearance that resembles pots used by Japanese fisherman to catch octopi known as “takotsubo.” While these patients typically undergo standard ACS treatment, ECG and ventricular function typically normalize within several months of the initial presentation. Importantly, the incidence of recurrence can be as high as 5% within 6 years.

In general, while “anxiety” and “panic disorder” will likely remain frequent diagnoses for ED patients with chest pain, EPs must remain cautious to not prematurely jump to these diagnoses before a thorough evaluation is performed.

## KEY POINTS

- Misdiagnosis of a cardiopulmonary condition as anxiety is a frequent occurrence.
- Patients with acute life-threatening cardiac or pulmonary conditions often present with feelings of panic or impending doom in association with chest pain.
- Moments of high stress and anger have been associated with an

increased incidence of cardiovascular events.

- Multiple studies have shown a link between anxiety disorders and higher rates of cardiac risk factors.
- Takotsubo cardiomyopathy is a reversible cardiomyopathy that occurs in the setting of a severe emotional event.

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## ONE AND DONE: RAPID RULE-OUT PROTOCOLS

MAITE ANNA HUIS IN 'T VELD, MD AND SEMHAR Z.  
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Chest pain is one of the most common patient complaints in the emergency department (ED). In order to prevent unnecessary increases in morbidity and mortality, it is imperative to rapidly identify patients with an acute coronary syndrome (ACS). Electrocardiography (ECG) abnormalities or elevated troponin values strongly suggest the presence of an ACS and the need for further evaluation. Many ED patients with an ACS, however, lack these characteristic abnormalities. Notwithstanding, it is expensive, potentially harmful, and simply not feasible to admit all ED patients who present with a complaint of chest pain. The challenge for the emergency provider (EP) is to identify chest pain patients at low risk of an ACS and who, subsequently, can be safely discharged from the ED.

Many authors have attempted to develop risk stratification systems to identify ED patients with acute chest pain that can be discharged. The most commonly referenced stratification systems are listed in [Table 63.1](#). Of the current systems, the HEART Score may be the most applicable tool for the EP and is the focus of the remainder of this chapter.

**TABLE 63.1 CHEST PAIN RISK SCORING SYSTEMS**

| <b>SCORE</b>                       | <b>POPULATION</b>   | <b>SCORE FACTORS</b>  | <b>OUTCOMES</b>  | <b>C-STATISTICS OF ORIGINAL STUDY</b> |
|------------------------------------|---|---|--|---------------------------------------|
| PURSUIT<br>(Boersma et al., 2000)  | Retrospective cohort of patients with unstable angina or NSTEMI       | Age<br>Sex<br>CCS* class<br>Signs of CHF<br>ST depression on initial ECG<br>Score 1–18  | Death, myocardial infarction at 30 d   | 0.84 (death), 0.67 (death/MI)         |
| TIMI<br>(Antman et al., 2000)      | Retrospective cohort of admitted patients with confirmed ACS          | Age<br>Risk factors<br>Known CAD<br>Aspirin use<br>Angina<br>Cardiac markers<br>ECG<br>Score 0–7  | All-cause mortality, AMI, severe recurrent ischemia requiring PCI after 14 d | 0.63                                  |
| GRACE<br>(Granger et al., 2003)    | Retrospective cohort of admitted patients with ACS (STEMI and NSTEMI) | Killip class<br>Systolic blood pressure<br>Heart rate<br>Age<br>Creatinine level<br>Other risk factors (including troponin, ECG, cardiac arrest on presentation)<br>Score 1–372 | In-hospital death and postdischarge death at 6 mo                            | 0.83                                  |
| FRISC<br>(Lagerqvist et al., 2004) | Retrospective cohort of admitted patients with unstable CAD or NSTEMI | Age<br>Sex<br>ECG<br>Risk factors<br>Troponin<br>Inflammatory markers<br>Score 0–7  | Death, AMI at 12 mo  | 0.77 (death), 0.70 (death/AMI)        |
| HEART<br>(Backus et al., 2008)     | Retrospective cohort of ED patients with chest pain                   | History<br>ECG<br>Age<br>Risk factors<br>Troponin<br>Score 0–10   | MACE, AMI, PCI or death at 6 wk  | 0.90                                  |

STEMI, ST-segment elevation myocardial infarction; NSTEMI, Non-ST Elevation MI; CHF, congestive heart failure; CAD, coronary artery disease.



The HEART Score was derived from a retrospective cohort of patients who presented to the ED with chest pain. These patients were followed for 6 weeks to measure the primary study end point of a major adverse cardiac event (MACE). MACEs were defined as acute myocardial infarction (AMI), primary coronary intervention (PCI), coronary artery bypass graft (CABG), or death. Importantly, the HEART Score is the only stratification system that incorporates the history of present illness (HPI) into the calculation. The remaining components of the HEART Score are listed in [Table 63.2](#). After the initial study, the authors conducted a large validation trial. These results are listed in [Table 63.3](#). A more recent validation study was conducted by Mahler et al., who compared the HEART Score to traditional care in ED patients with chest pain. In this study, the HEART Score decreased cardiac testing at 30 days by 12.1%, ED length of stay by 12 hours, and increased early ED discharge by 21.3%. There were no MACEs reported in the early discharge group at 30 days.

**TABLE 63.2 HEART SCORE**

|              |                                    |          |
|--------------|------------------------------------|----------|
| History      | Highly suspicious                  | 2 points |
|              | Moderately suspicious              | 1 point  |
|              | Slightly or nonsuspicious          | 0 points |
| ECG          | Significant ST depression          | 2 points |
|              | Nonspecific repolarization         | 1 point  |
|              | Normal                             | 0 points |
| Age          | ≥65 years                          | 2 points |
|              | >45–<65 years                      | 1 point  |
|              | ≤45 years                          | 0 points |
| Risk factors | ≥3 risk factors or history of CAD  | 2 points |
|              | 1 or 2 risk factors                | 1 point  |
|              | No risk factors                    | 0 points |
| Troponin     | ≥3× normal limit                   | 2 points |
|              | >1–<3 normal limit                 | 1 point  |
|              | Less than or equal to normal limit | 0 points |

**TABLE 63.3 RESULTS OF INITIAL HEART STUDY AND VALIDATION STUDY**

|                                   | <b>RISK OF MACE<br/>AT 6 WK (BACKUS,<br/>2008)</b> | <b>RISK OF MACE AT<br/>6 WK (BACKUS,<br/>2013)</b> |
|-----------------------------------|--|--|
| Low HEART Score<br>(0–3)          | 2.5% (1/39)  | 1.7% (15/870)                                      |
| Intermediate HEART<br>Score (4–6) | 20.3% (12/59)                                      | 16.6% (183/1101)                                   |
| High HEART Score<br>(7–10)        | 72.7% (16/22)                                      | 50.1% (209/417)                                    |

There are limitations to the HEART Score. The most notable is the subjective component of the HPI. Another limitation is validation. Though there have been several validation studies performed, there are currently no studies that are adequately powered to detect a difference in MACE. Furthermore, the validation study by Backus et al. reported a nonadherence rate of 29% to the HEART Score in patients considered low risk for an ACS.

These scoring systems can be used to assist the EP in identification of ED patients that can be safely discharged. The HEART Score has been shown to be a reliable and valuable tool for low-risk ED chest pain patients. Notwithstanding, future studies are needed to further validate the use of the HEART Score in a larger group of patients.

## KEY POINTS

- Multiple risk scoring systems are available for evaluation of acute chest pain.
- The HEART Score is the only tool that was developed to assess ED chest pain patients.
- The HEART Score incorporates the patient's HPI, ECG, age, risk factors for coronary artery disease, and troponin value.
- Patients with a HEART Score <3 have a <2% risk of MACE at 6 months.

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## BEWARE OF THE “HIGHLY SENSITIVE” TROPONIN

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Acute chest pain accounts for ~5 million emergency department (ED) visits annually in the United States (US). Cardiac enzymes are routinely used to risk stratify ED patients with acute chest pain when there is concern for an acute myocardial infarction (AMI) or acute coronary syndrome (ACS). Troponins T and I are the current standard cardiac enzymes for the diagnosis of AMI, with a sensitivity of ~85% for cardiac injury. Importantly, current troponin assays may take several hours after the onset of AMI symptoms to return abnormal. It is for this reason that many current ED chest pain protocols obtain serial troponin values over the course of several hours to exclude an AMI.

In 2009, “highly sensitive” troponin assays became available. These assays can detect the presence of troponin at much lower serum levels compared to traditional troponin assays. It is important to understand what is meant by the term “highly sensitive.” For a troponin assay to be labeled as “highly sensitive,” the following two criteria must be met:

- 1) The assay has to detect troponin in more than 50% of healthy patients. Traditional assays do not detect troponin in healthy individuals who do not have myocardial disease; therefore, any elevation in a traditional troponin value is abnormal. This is not the case with “highly sensitive” troponin values.
- 2) The analytic precision has to be high. Precision is measured with the coefficient of variation (CV). The CV is calculated by performing the same test on the same blood sample several different times. The



|            |  |
|------------|--|
| Cardiac    | Acute coronary syndrome<br>Dysrhythmias<br>Congestive heart failure<br>Peri-/myocarditis<br>Aortic valve disease<br>Cardiac contusion<br>Infiltrative cardiac disorders<br>Postcardiac procedure |
| Noncardiac | Pulmonary embolism<br>Aortic dissection<br>End-stage renal disease<br>Sepsis<br>Rhabdomyolysis   |

## WHAT DOES A NEGATIVE RESULT MEAN?

Initial studies on “highly sensitive” troponins reported a negative predictive value for AMI between 99% and 100%. Recent studies have failed to replicate these initial findings. A recent international study found that up to 23% of patients ultimately diagnosed with an AMI had an initial negative “highly sensitive” troponin. Sensitivity and negative predictive value, however, did improve when troponin values were obtained 6 or more hours after symptom onset.

Troponin values should be used as a risk stratification tool. It is imperative to recognize that a negative value does not exclude AMI, whereas a positive troponin value is not always due to an AMI. The current literature does not support the use of “highly sensitive” troponins as a single modality to exclude or diagnose AMI. In many cases, repeat values are needed, similar to the current utilization of traditional troponin assays. At present, the U.S. Food and Drug Administration has not approved “highly sensitive” troponin assays for use in clinical practice.

### KEY POINTS

- Troponin values should be used to risk stratify patients with acute

chest pain.

- There are numerous noncardiac etiologies for an elevated troponin value.
- “Highly sensitive” troponins have a higher sensitivity and negative predictive value, but lower specificity, when compared with traditional troponin assays.
- Up to 23% of patients with an AMI may have an initial negative “highly sensitive” troponin value.
- There are insufficient data to support the use of a single “highly sensitive” troponin to exclude AMI.

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## WHEN GOOD VADs GO BAD

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**CHRISTINA LYNN TUPE, MD, RDMS**

With the development of the ventricular assist device (VAD), patients with end-stage cardiac failure now have an increased survival rate and improved quality of life. There are currently three indications for placement of a VAD: as a bridge to recovery in patients whose cardiac function is temporarily diminished (i.e., myocarditis), as a bridge to cardiac transplantation, and as destination therapy for patients who are not candidates for cardiac transplantation. The left ventricular assist device (LVAD) is the most common VAD in clinical practice, though a right ventricular assist device (RVAD) and a biventricular assist device (BiVAD) are also available. A well-coordinated multidisciplinary team manages VAD patients. Notwithstanding, these patients will develop complications from the device that require emergency department (ED) evaluation and treatment. As such, it is imperative for the emergency provider (EP) to have a systematic approach to the evaluation of these complex patients.

The ED evaluation of a VAD patient should begin with assessment of the patient's airway, breathing, and circulation. Current VA devices deliver a continuous flow of blood to the patient. As a result, a pulse is often absent or markedly diminished. Noninvasive systolic and diastolic blood pressure measurements may be inaccurate or simply not obtainable. For VAD patients, the mean arterial blood pressure (MAP) can be obtained with a blood pressure cuff and Doppler ultrasound over the brachial or radial artery. The cuff should be inflated until flow ceases on the Doppler device. The cuff should then be slowly deflated. Auscultation of the first flow signal indicates the patient's MAP. The target range for MAP in VAD patients is 70 to 90 mm Hg. An arterial line should be considered in the VAD patient who appears critical or moribund. It is also important to evaluate additional signs of peripheral perfusion, such as mental status, skin color and warmth, and urine output. The patient's chest should be auscultated to determine the



presence, or absence, of the continuous hum of the VAD. The absence of an audible hum suggests catastrophic VAD dysfunction and the need for immediate resuscitation.

Additional components of the VAD that should be included in the initial assessment are the speed, flow, power, and battery life of the device. These values are found on the VAD monitor. Once the initial assessment has occurred, the patient's VAD coordinator should be contacted to assist with management.

Atrial or ventricular dysrhythmias can occur in up to 50% of VAD patients. As such, it is important to obtain an electrocardiogram (ECG) in most VAD patients. Common etiologies for dysrhythmias include hypovolemia, electrolyte derangements, and myocardial ischemia. VAD patients who are unstable due to a dysrhythmia should be cardioverted or defibrillated. It is recommended to place the defibrillation pads in an anterior and posterior position. VAD patients who are stable yet have a concerning dysrhythmia can receive antiarrhythmic medications. These patients should also receive intravenous fluids and, if necessary, electrolyte replacement.

Bedside echocardiography (echo) is an invaluable tool in the ED evaluation of VAD patients, especially those who appear critically ill. Echo can be used to evaluate for pump thrombosis, right ventricular (RV) failure, and "suction" events. Pump thrombus occurs in up to 2% of patients within 2 years after VAD implantation. Pump thrombosis reduces cardiac output, which results in an elevation in the pump power reading. VAD patients with suspected thrombosis should receive anticoagulation with heparin. Acute RV failure can occur in up to 25% of VAD patients and is usually seen soon after implantation. VADs are preload-sensitive devices. In low-flow states, the negative pressure produced by the VAD can cause leftward displacement of the intraventricular septum and produce a "suction" event. Suction events usually result from hypovolemia, but they can also occur with cardiac tamponade, dysrhythmias, and malposition of the inflow cannula. Initial management of a suction event includes intravenous fluids, echo, and arrangement for transfer to a VAD center to evaluate the inflow cannula.

Bleeding is an important and common complication that occurs in up to 40% of VAD patients. All VAD patients are placed on anticoagulant and antiplatelet medications to decrease the rate of thromboembolic events. In addition, these patients develop an acquired von Willebrand syndrome in response to shear forces from the VAD. Finally, the decreased pulse pressure of the continuous flow contributes to arteriovenous malformations, especially in the jejunum. VAD patients who are unstable due to hemorrhage should receive blood products and agents to reverse anticoagulation.

Anticoagulation reversal in a stable VAD patient, however, should be done in consultation with the patient's VAD team.

VAD patients are at high risk for infection. Infection can occur anywhere along the VAD including the surgical site, driveline, pump, or device pocket. VAD infections can be caused by a variety of organisms, including gram-positive organisms, especially coagulase-negative staphylococci and *Staphylococcus aureus*, gram-negative organisms, and fungi. Critically ill VAD patients should receive broad-spectrum antibiotics to cover both gram-positive and gram-negative organisms.

Rarely, VAD patients may present in cardiac arrest. In these patients, the controller should be quickly evaluated for battery life and proper connections. Echo should be used to evaluate for pericardial effusion, left ventricular (LV) function, or RV dilatation. Most VAD manufacturers state that cardiopulmonary resuscitation (CPR) should be done only if absolutely necessary, primarily due to concern of dislodgement of the inflow cannula from the LV. The only literature that exists on CPR for the VAD patient is a recent retrospective analysis of eight VAD patients who received CPR. In this study, no patient experienced dislodgement of the device with a 50% survival rate with good neurologic outcome.

VAD patients are a special challenge for the EP. Through a careful assessment of the VAD, physical exam, MAP, ECG, and echo, the EP can resuscitate the good VAD that has gone bad.

## KEY POINTS

- Contact the patient's VAD coordinator as soon as possible.
- Obtain an ECG early to evaluate for dysrhythmias.
- Consider bleeding and sepsis in any critically ill VAD patient.
- Use echo to assess for pump thrombosis, RV failure, and suction events in the VAD patient.
- CPR is reasonable in the VAD patient in cardiac arrest.

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# DON'T STRESS THE STRESS TEST IN SUSPECTED ACS

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**CHRISTINA LYNN TUPE, MD, RDMS**

The evaluation of the emergency department (ED) patient with acute chest pain centers on the history of present illness (HPI), physical examination, and electrocardiogram (ECG). Often, the emergency provider (EP) utilizes troponin values and the results of prior cardiac stress tests to further risk stratify patients and determine the need for additional management. The role of prior cardiac stress tests in the risk stratification of ED patients with suspected acute coronary syndrome (ACS) is uncertain.

The goal of a cardiac stress test is to identify the patient with obstructive coronary artery disease (CAD), typically defined as stenosis of 50% or more of a coronary artery on angiography. Traditional stress tests include exercise ECG (on a treadmill), echocardiography, or nuclear medicine studies. All have rest and exercise phases. The exercise phases of echocardiography and nuclear medicine studies can be performed with physical exertion or chemically induced with medication such as adenosine or dobutamine. Depending on the study, the test will detect ischemic ECG changes, wall motion abnormalities, or diminished tracer uptake indicative of decreased perfusion to regions of the myocardium. Test availability, the patient, and the presence of comorbid conditions often determine the type of stress test that is performed.

The pooled sensitivity for all cardiac stress tests range from 67% to 85%, with specificity that ranges from 70% to 95%. Stress tests have higher sensitivity in patients with multivessel CAD. Exercise ECG stress tests have a sensitivity of 68% and a specificity of 77% for CAD. The presence of multivessel disease increases the sensitivity to 81%. Importantly, the sensitivity and specificity are lower in women than in men. Most

cardiologists obtain an exercise ECG stress test as the initial outpatient test. Recently, coronary computed tomography angiography (CCTA) has emerged as a means of diagnosing CAD and defining coronary lesions. The pooled sensitivity of CCTA ranges between 98% and 99%, with a specificity between 82% and 89%. The positive predictive value of CCTA is reported to be 85%, whereas the negative predictive value is ~92%.

The ED patient with acute chest pain is very different from the asymptomatic patient who undergoes a routine outpatient cardiac stress test. Nerenberg and colleagues reviewed the disposition of ED patients evaluated for ACS. They authors found that a previous negative stress test did not change the rate of hospital admission. Furthermore, there was no difference in the rate of adverse events among patients with a positive stress test, a negative stress test, or no previous stress test. In another series of ED patients, Smith and associates found that ~5% of patients were diagnosed with an acute myocardial infarction within 3 years of a negative stress test result. Walker and colleagues reviewed the records of ED patients with chest pain and a negative, or inconclusive, stress test result within the preceding 3 years. This study included treadmill and pharmacologic echocardiograms, pharmacologic nuclear medicine studies, treadmill nuclear studies, and a treadmill ECG-only evaluation. The authors defined CAD as a myocardial infarction identified by positive cardiac enzymes, a subsequent positive stress test, a cardiac catheterization that required intervention, coronary artery bypass graft surgery, or death caused by cardiac arrest. Approximately 20% of patients were diagnosed with CAD after a negative stress test within 3 years of the ED presentation. Of patients with significant CAD, 23.5% had a negative stress test *within 1 month* before their ED presentation.

Cardiac stress test is a valuable tool for cardiologists to screen outpatients for CAD. In an outpatient setting, stress tests are used to identify fixed obstructions to coronary artery flow. In ED patients with acute chest pain, however, the intent of testing is to identify acute plaque rupture and thrombus formation. As such, cardiac stress tests have limited utility in the ED evaluation of acute chest pain. The EP should not exclude an ACS in the ED patient with acute chest pain based solely on a recent negative stress test result. If the HPI is concerning, the EP should continue with the evaluation and subsequent admission.

## KEY POINTS

- Cardiac stress tests have a pooled sensitivity of 67% to 85%.

- The sensitivity of stress tests is higher in patients with multivessel disease than in those with single-vessel disease.
- The sensitivity and specificity of exercise ECG stress testing are lower in women than in men.
- A previous negative stress test should not be the basis for subsequent decisions regarding hospital admission.
- Significant CAD can be present despite a recent negative stress test result.

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## **REMEMBER TO OBTAIN A RIGHT-SIDED ELECTROCARDIOGRAM IN A PATIENT WITH AN INFERIOR MYOCARDIAL INFARCTION**

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**CARMEN AVENDANO, MD AND SEMHAR Z. TEWELDE, MD**

An acute inferior myocardial infarction (IMI) typically causes ST-segment elevation (STE) in leads II, III, and aVF on the 12-lead electrocardiogram (ECG). In addition to these findings, an IMI can also produce ST-segment depression (STD) or T-wave inversion (TWI) in lead aVL. In fact, STD or a new TWI in lead aVL is often the first ECG abnormality seen in patients with an acute IMI. This underscores the importance of obtaining serial ECGs in patients with a suspected acute coronary syndrome.

When an acute IMI is diagnosed, a right-sided ECG should be obtained to exclude the presence of a concomitant right ventricular myocardial infarction (RVMI). RVMIs can complicate 30% to 50% of acute IMIs. A right-sided ECG is performed by taking leads V3 through V6 and placing them on the right side of the chest. These leads are then labeled V3R through V6R. STE of 0.5 mm or greater in one or more of leads V4R through V6R has a sensitivity of 90% and specificity of 91% for an RVMI. In an RVMI, STE is more commonly seen in lead V4R compared with leads V5R and V6R. In fact, STE greater than 1 mm in lead V4R suggests a proximal occlusion of the right coronary artery (RCA) and an increased risk for atrioventricular block. Additional ECG findings that suggest an RVMI include STE in lead V1, STE in lead III that is greater than the STE in lead

II, and an isoelectric ST segment in lead V1 with STD in lead V2.

It is important to diagnose an RVMI because it affects patient management. Patients with an acute myocardial infarction commonly receive nitrate medications in addition to other time-sensitive therapies. Patients with an RVMI are preload dependent and require intravenous fluids to maintain adequate perfusion. A precipitous decline in blood pressure can occur when nitrates are administered to patients with an RVMI. Any medication that may decrease preload should be avoided in the setting of an RVMI. Morphine has been shown to increase infarct size and decrease coronary blood flow by up to 13% in patients with an RVMI. Morphine should be avoided in patients with an RVMI. Additional therapies for patients with an RVMI remain the same as those for patients with non-RVMIs.

## KEY POINTS

- RVMI can complicate up to 50% of IMIs.
- When an IMI is identified, a right-sided ECG should be obtained.
- STE in lead V4R indicates a proximal occlusion of the RCA and an increased risk for AVB.
- STE in lead V1 with concomitant depression in lead V2 suggests an RVMI.
- Patients with an RVMI are preload dependent. Avoid nitrates and administer intravenous fluids.

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# PITFALLS IN HYPERTENSIVE EMERGENCIES

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STEPHEN D. LEE, MD

Approximately 1% to 2% of patients with hypertension will present with a hypertensive emergency, defined as organ dysfunction due to an elevated blood pressure. Importantly, there is no specific blood pressure threshold that identifies a patient with a hypertensive emergency. In a hypertensive emergency, the initial pathophysiologic event is an abrupt increase in systemic vascular resistance (SVR). The abrupt increase in SVR causes endothelial injury and results in increased vascular permeability, platelet activation, and fibrin deposition. This fibrin deposition causes microvascular thrombi, vessel occlusion, organ ischemia, and ultimately organ dysfunction.

Patients with a hypertensive emergency should be treated with an intravenous vasodilator medication. While patients with asymptomatic hypertension can be safely treated with oral medications to slowly reduce blood pressure, patients with organ dysfunction due to elevated blood pressure should be given an easily titratable intravenous medication. This allows for a safe, controlled, and appropriate reduction in blood pressure in order to halt organ dysfunction.

Appropriate reduction in blood pressure is central to the management of a hypertensive emergency. The target reduction in blood pressure depends on the disease process and the specific organ involved. An abrupt and aggressive reduction in blood pressure can cause further injury and ischemia, as blood pressure can drop below the patient's autoregulatory threshold. Current literature recommends that the mean arterial blood pressure (MAP) be lowered no more than 25% in the first 1 to 2 hours from the time of diagnosis. [Table 68.1](#) lists common hypertensive emergencies and the current recommended blood pressure targets. Conditions that require more

aggressive MAP reduction in a shorter time period include aortic dissection, intracerebral hemorrhage, and eclampsia. For these conditions, it may be necessary to reduce MAP more than 25% in the first 2 hours in order to minimize progressive organ injury. In contrast, it may be preferable to avoid blood pressure reduction altogether for the patient with an acute ischemic stroke, except in those patients with severe hypertension (>220/110 mm Hg) or those receiving thrombolysis (>185/110 mm Hg). For patients with acute myocardial infarction or acute pulmonary edema, MAP is reduced until clinical symptoms improve.

**TABLE 68.1 HYPERTENSIVE EMERGENCIES AND BLOOD PRESSURE GOALS**

| <b>HYPERTENSIVE EMERGENCY</b>   | <b>TREATMENT GOALS</b>                       |
|---------------------------------|--|
| Ischemic stroke                 | <220/120 mm Hg                               |
| Ischemic stroke w/ thrombolysis | <185/110 mm Hg                               |
| Intracerebral hemorrhage        | <160/90 mm Hg or MAP < 110 mm Hg             |
| Aortic dissection               | SBP 100–120 mm Hg, HR < 60 bpm               |
| Heart failure/pulmonary edema   | Symptom relief (often with 10%–15% decrease) |
| (Pre)eclampsia                  | <160/90 mm Hg                                |

There is currently limited data on the medications used to treat patients with a hypertensive emergency. Medications are best chosen according to the disease process. The most commonly used medications include calcium channel blockers (e.g., nicardipine) and beta-blockers (e.g., labetalol). Nicardipine is a second-generation calcium channel blocker that results in coronary and cerebral vasodilation. Nicardipine is commonly used to treat patients with a neurologic emergency, as it has little effect on intracranial pressure. In contrast, sodium nitroprusside is an arterial and venous vasodilator that can cause a coronary and cerebral “steal” phenomenon. Nitroprusside should thus be avoided in patients with ischemic stroke or acute myocardial infarction. Nitroprusside is also best avoided in patients with eclampsia, as its by-product (cyanide) can accumulate with prolonged use and may cause harm to the fetus. Beta-blockers (e.g., esmolol) should be the first-line treatment for patients with an aortic dissection to reduce heart rate. Once the target heart rate is reached, another vasodilator (e.g., nicardipine) can be administered to reduce the systolic blood pressure (SBP) to 100 to 120 mm Hg. The direct vasodilator hydralazine should be avoided in patients with a hypertensive emergency, as it can cause a reflex tachycardia and has a duration of action that can exceed 10 hours.

It is important to consider secondary causes of hypertension in the patient with a hypertensive emergency. Secondary causes of hypertension include endocrine conditions (e.g., pheochromocytoma), drugs of abuse (e.g., cocaine, amphetamines, phencyclidine), and medication or drug withdrawal syndromes. In these conditions, beta-blocker medications should be avoided to prevent a catecholamine surge from unopposed alpha-adrenergic receptor stimulation. The addition of other medications, such as a benzodiazepine or clonidine, may prove beneficial in treating patients with these hyperadrenergic states.

## KEY POINTS

- Hypertensive emergency is organ dysfunction due to elevated blood pressure.
- Lower the MAP by no more than 25% in the first 2 hours to avoid hypoperfusion and organ ischemia.
- Tailor your pharmacologic agent to the disease process.
- Avoid reflex tachycardia in aortic dissection by using a beta-blocker first.
- Consider alternative sources for elevated blood pressure (e.g., cocaine, pheochromocytoma).

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# KNOW THE DIFFERENTIAL FOR ST-SEGMENT ELEVATION: IT'S MORE THAN JUST ACUTE CORONARY SYNDROME

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**KATHLEEN STEPHANOS, MD AND SEMHAR Z. TEWELDE, MD**

Emergency providers (EPs) interpret electrocardiograms (ECGs) on a daily basis for a variety of clinical scenarios. An ECG is most commonly obtained for the emergency department (ED) patient with acute chest pain. In these patients, it is imperative to diagnose an ST-segment elevation myocardial infarction (STEMI), as emergent reperfusion therapy is indicated. Notwithstanding, there are numerous conditions that cause ST-segment elevation (STE) on an ECG that are not attributable to an acute coronary syndrome. In order to avoid errors in diagnosis or management, it is important for the EP to know the differential diagnosis for STE.

Early repolarization is a common cause of STE. It is typically seen in males younger than 45 years of age and in athletic patients. Recent literature has demonstrated that early repolarization is a risk factor for sudden cardiac death, as it increases the risk for idiopathic ventricular fibrillation. Early repolarization typically produces a notched J-point with STE that is <3 mm on the ECG. There is currently no specific treatment for early repolarization. Patients should be instructed to follow up with their primary care physician. Cardiology referral is appropriate for patients who have a personal or family history of sudden cardiac death or additional ECG findings that suggest the presence of coronary artery disease.

Pericarditis is another condition that should be considered in the differential diagnosis of STE. Patients typically present with acute chest pain that is pleuritic and positional. Importantly, pericarditis produces diffuse STE that is concave in appearance. The STE is most prominent in ECG leads I, II, III, aVF, aVL, and V2 to V6. The presence of reciprocal ST-segment depression or T-wave inversions is more consistent with the diagnosis of STEMI. In addition, STE that is flat or horizontal should be considered an STEMI until proven otherwise. PR-segment depression is commonly thought to be pathognomonic for pericarditis; however, this finding can be transient and can also be seen in patients with an STEMI. Rapid evolution of ST-segment changes during the course of the ED evaluation is more suggestive of an acute coronary syndrome in contrast to pericarditis. The ECG changes seen in pericarditis typically occur over the course of several weeks.

Left bundle-branch block (LBBB), left ventricular hypertrophy (LVH), and left ventricular aneurysm can all cause STE. In the setting of LBBB, the ST segment is directly opposite to that of the main QRS complex vector. In leads V1 to V3, the QRS vector is negative, thereby producing STE. This discordant STE is typically less than 5 mm in the setting of LBBB. Patients with a pacemaker will also demonstrate STE similar to a LBBB. LVH produces discordant STE, with a deep S wave in leads V1 to V3 and a tall R wave in leads I, aVL, V5, and V6. Patients with a left ventricular aneurysm may have STE along with deep anterior or septal Q waves in leads V1 to V3. Similar to pericarditis, LBBB, LVH, and left ventricular aneurysm do not cause dynamic ECG abnormalities.

Hyperkalemia is often referred to as the “great imitator” of ECG abnormalities. Hyperkalemia can produce numerous ECG findings that can easily be misdiagnosed and incorrectly treated. It is well known to cause the appearance of STE, as the T-wave is pulled to a peak. Additional signs of hyperkalemia include peaked T-waves, bradydysrhythmias, tachydysrhythmias, and widened QRS complexes.

Less common causes of STE include Prinzmetal angina and takotsubo cardiomyopathy. Both of these clinical entities are often diagnosed as STEMI in patients with acute chest pain. These diagnoses are often made following emergent cardiac catheterization.

## KEY POINTS

- STEMI is not the only cause of STE.
- Dynamic ECG abnormalities are more consistent with an acute

coronary syndrome.

- Nonischemic causes of STE include early repolarization, LBBB, LVH, pericarditis, and left ventricular aneurysm.
- Hyperkalemia should be considered in the differential diagnosis of STE.
- The diagnosis of Prinzmetal angina or takotsubo cardiomyopathy is often made following emergent cardiac catheterization for presumed STEMI.

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# DO NOT RELY ON A SINGLE ECG TO EVALUATE CHEST PAIN IN THE ED

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**KATHLEEN STEPHANOS, MD AND SEMHAR Z. TEWELDE, MD**

Chest pain is the second most common emergency department (ED) complaint and accounts for over 8 million ED visits annually in the United States. Fortunately, only a minority of patients will have a life-threatening cause of their acute chest pain. Notwithstanding, it is critical to identify patients with an acute coronary syndrome (ACS). At present, ~2% of ED patients with an ACS are misdiagnosed, which leads to increased morbidity and mortality.

The electrocardiogram (ECG) is a quintessential component of the evaluation of ED patients with acute chest pain. It is one of the most commonly utilized diagnostic tests in emergency medicine. Significant ST-segment elevation with reciprocal changes in the patient with acute chest pain is often easily recognized as an ACS. However, the initial ECG frequently demonstrates nonspecific changes, or is normal, in the setting of an ACS. As a result, it is important for the emergency provider to recognize the limitations of a single ECG in the evaluation of ED patients with acute chest pain.

Up to 20% of chest pain patients who ultimately require reperfusion therapy have an initial ECG that is normal or displays nonspecific abnormalities. Ischemic changes can occur rapidly and unbeknownst to the clinician if an ECG is not repeated. Current guidelines from the American Heart Association for the evaluation of low-risk ED patients with chest pain

recommend serial ECGs. Serial ECGs are also recommended in the most recent AHA guideline for non–ST-segment elevation myocardial infarction. Serial ECGs improve the sensitivity of identifying an ACS from 43% to 83%. Ideally, an ECG should be repeated every 5 to 10 minutes in symptomatic patients or those who have a change in the character of their chest pain. While this short time frame for a repeat ECG can be challenging to meet in a busy ED, it can make a significant impact upon the delivery of time-sensitive therapies. The initial, and repeat, ECGs should be compared with any prior ECGs to identify subtle abnormalities such as new T-wave inversions.

Currently, it is unclear how many ECGs should be obtained in the ED patient with acute chest pain. Nevertheless, it is a high-yield diagnostic test with minimal cost. For select patients, serial ECGs may make the difference between life and death due to ACS.

## KEY POINTS

- Approximately 2% of patients with an ACS are missed.
- The initial ECG in patients with an ACS is often nonspecific.
- Serial ECGs improve the accuracy of an ACS diagnosis.
- In symptomatic patients, obtain serial ECGs every 5 to 10 minutes.
- Compare the current ECG to prior ECGs to detect subtle changes.

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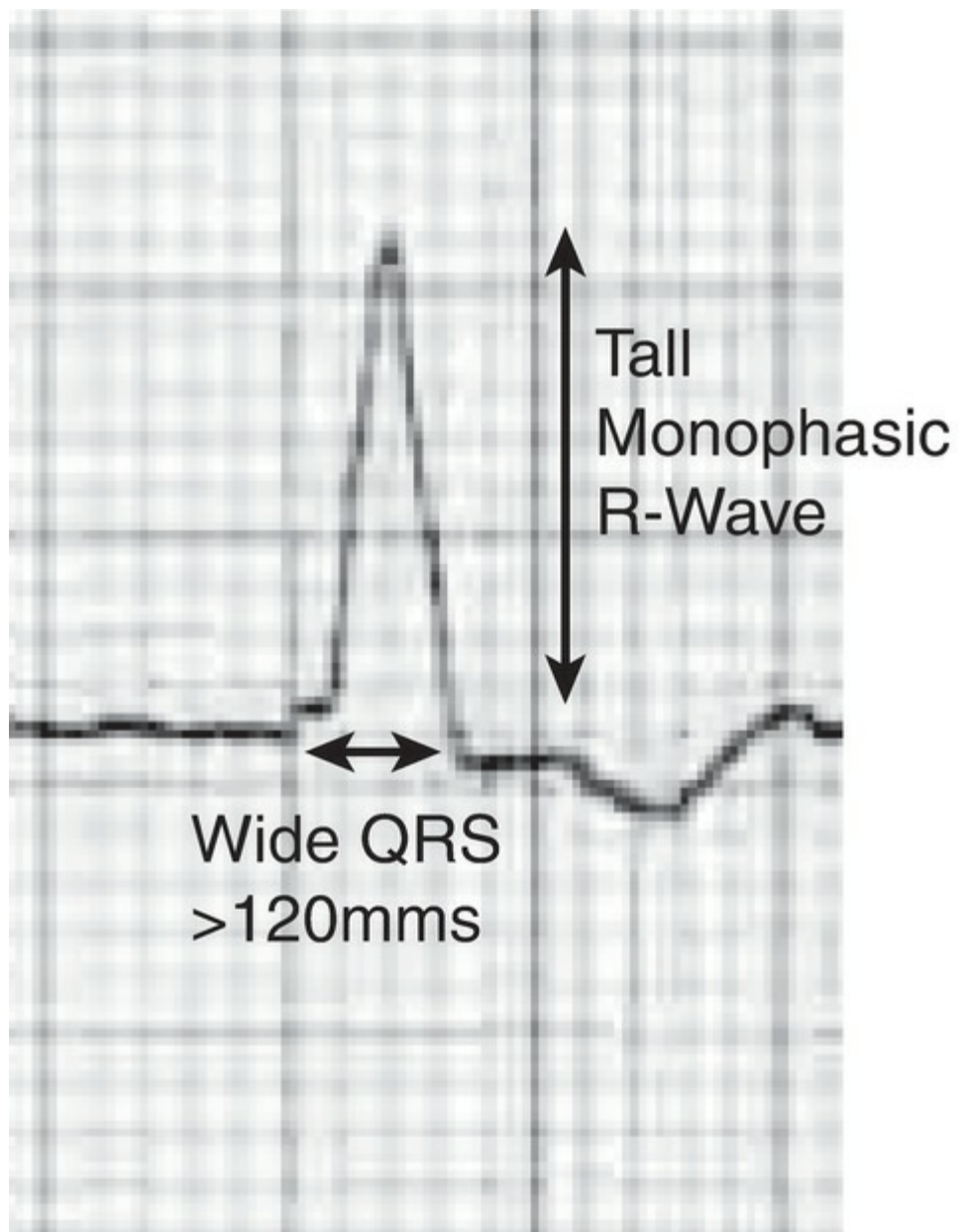


# KNOW HOW TO DIAGNOSE ACUTE MI IN PATIENTS WITH AN LBBB OR PACEMAKER

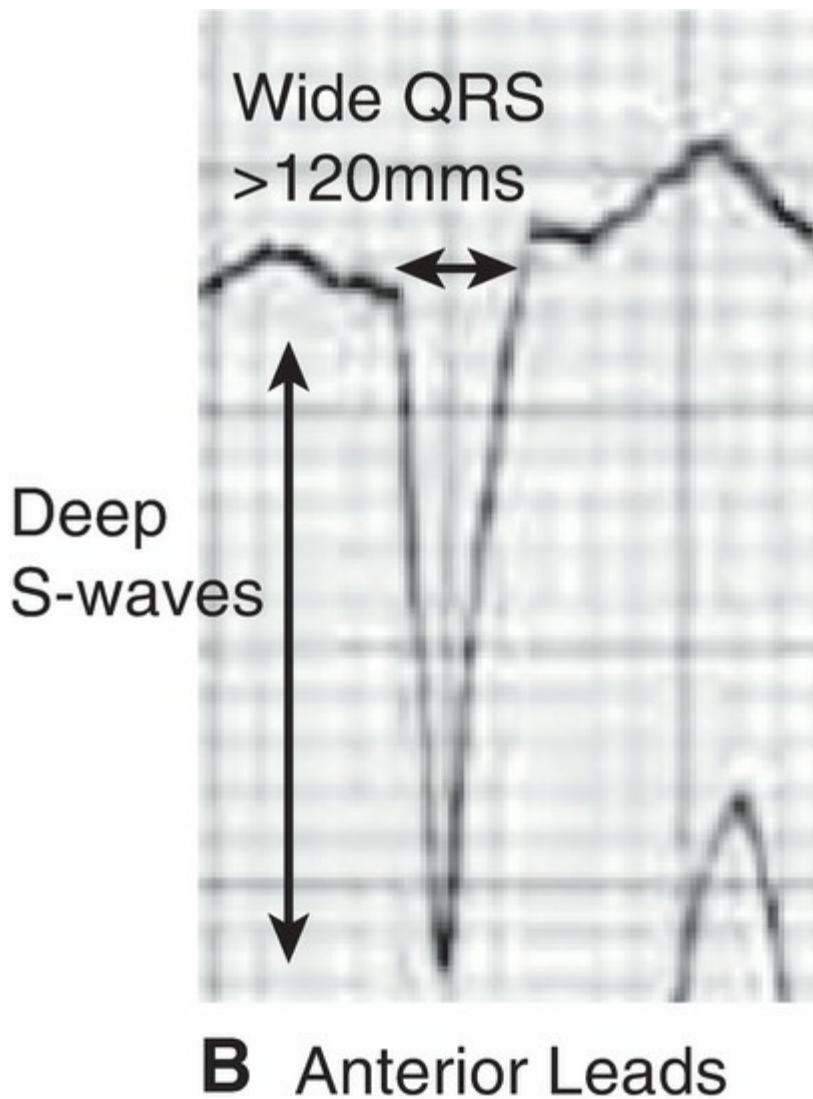
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**ANTHONY ROGGIO, MD**

A left bundle branch block (LBBB) is generated on an electrocardiogram (ECG) when an impulse is impeded as it passes down the conduction system and attempts to innervate the left side of the heart. As a result, cardiac myocytes are initially depolarized through the right bundle and then pass through the septum to depolarize the left ventricle. This results in sequential depolarization of the ventricles from right to left and creates an ECG pattern characterized by a QRS complex  $>120$  ms, a deep S wave in the anterior leads V1 to V3, and a tall monophasic R wave in leads I, V5, and V6 ([Figure 71.1](#)). A similar ECG pattern can be seen in patients with a ventricular pacemaker. The pacemaker leads first activate the myocytes of the right ventricle, followed by depolarization of the left ventricle myocytes.



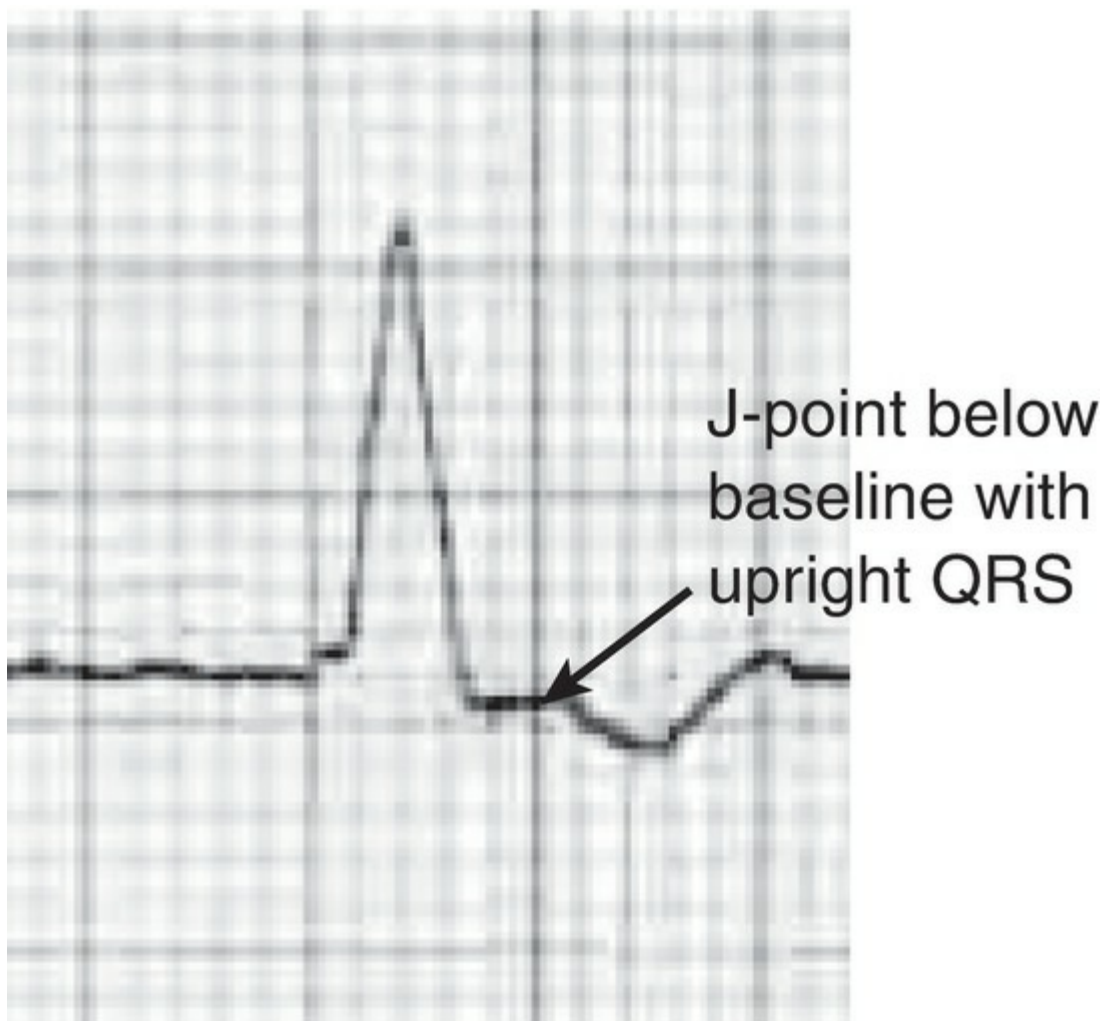
**A** Inferior Leads



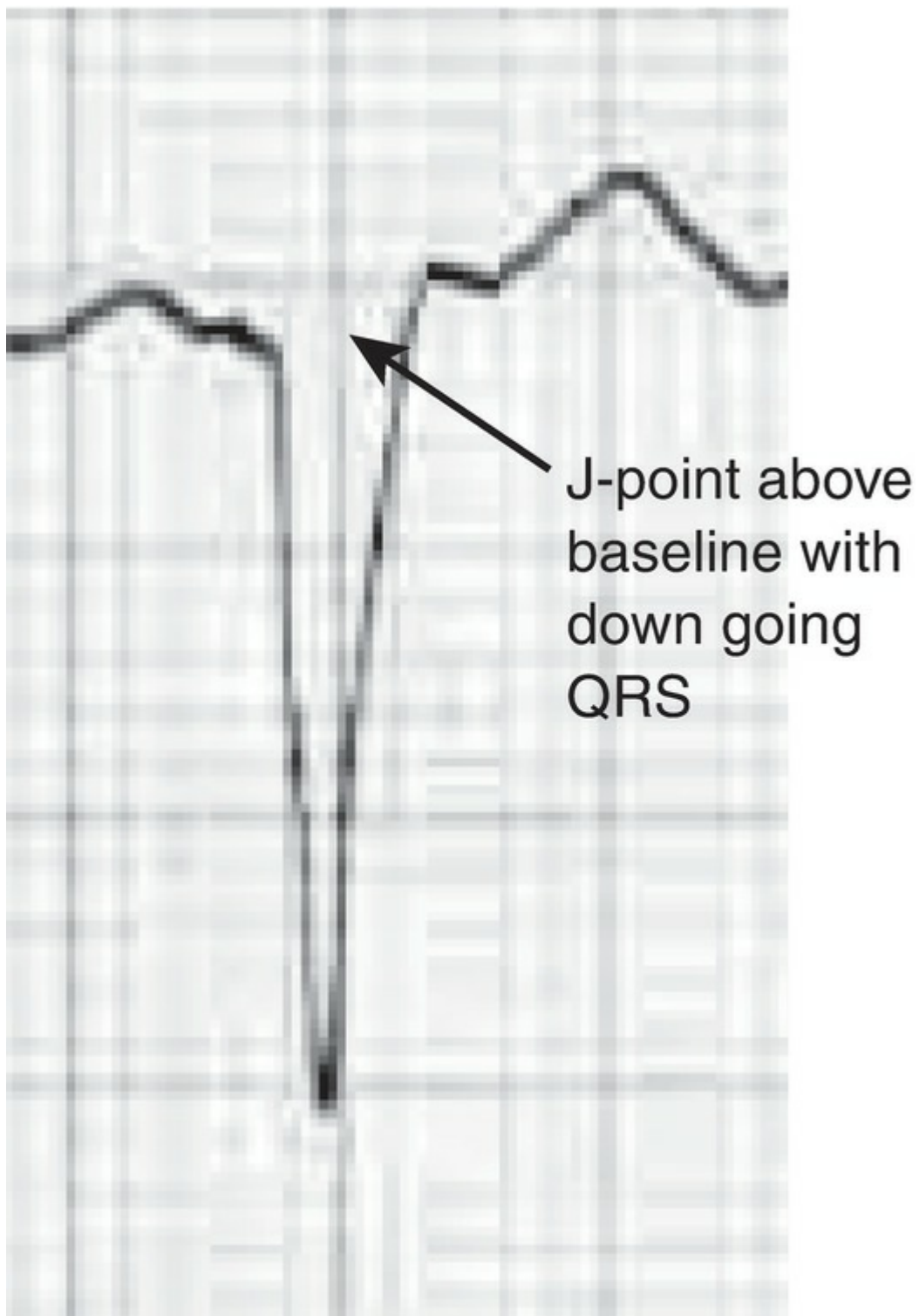
**Figure 71.1** Left bundle branch block defining characteristics. Image from Creative Commons, Left bundle branch block (LBBB). 2015.

In LBBB (with or without a ventricular pacemaker), there is discordance between the QRS complex and the J-point. When the QRS complexes point upward, the J-point should be below the isoelectric line. Conversely, when the QRS complex points downward, the J-point should be above the isoelectric line. This phenomenon is known as “appropriate discordance” ([Figure 71.2](#)) and leads to difficulty in the diagnosis of an acute ST-segment elevation myocardial infarction (STEMI). In fact, classic teaching states that an acute STEMI cannot be diagnosed on ECG in the presence of an LBBB. This has led to the common practice of administering emergent reperfusion therapy (thrombolytic medication or cardiac catheterization) in patients who have a new LBBB on ECG and symptoms that are consistent with a myocardial infarction. However, recent literature has questioned the benefit of emergent reperfusion therapy in patients with a new LBBB, where up to 86% of patients with an LBBB do not have an acute coronary artery

occlusion. As a result of recent literature, the finding of a new, or presumed new, LBBB on the ECG has been removed from current guidelines as an indication for emergent reperfusion therapy.



**A** Inferior Leads

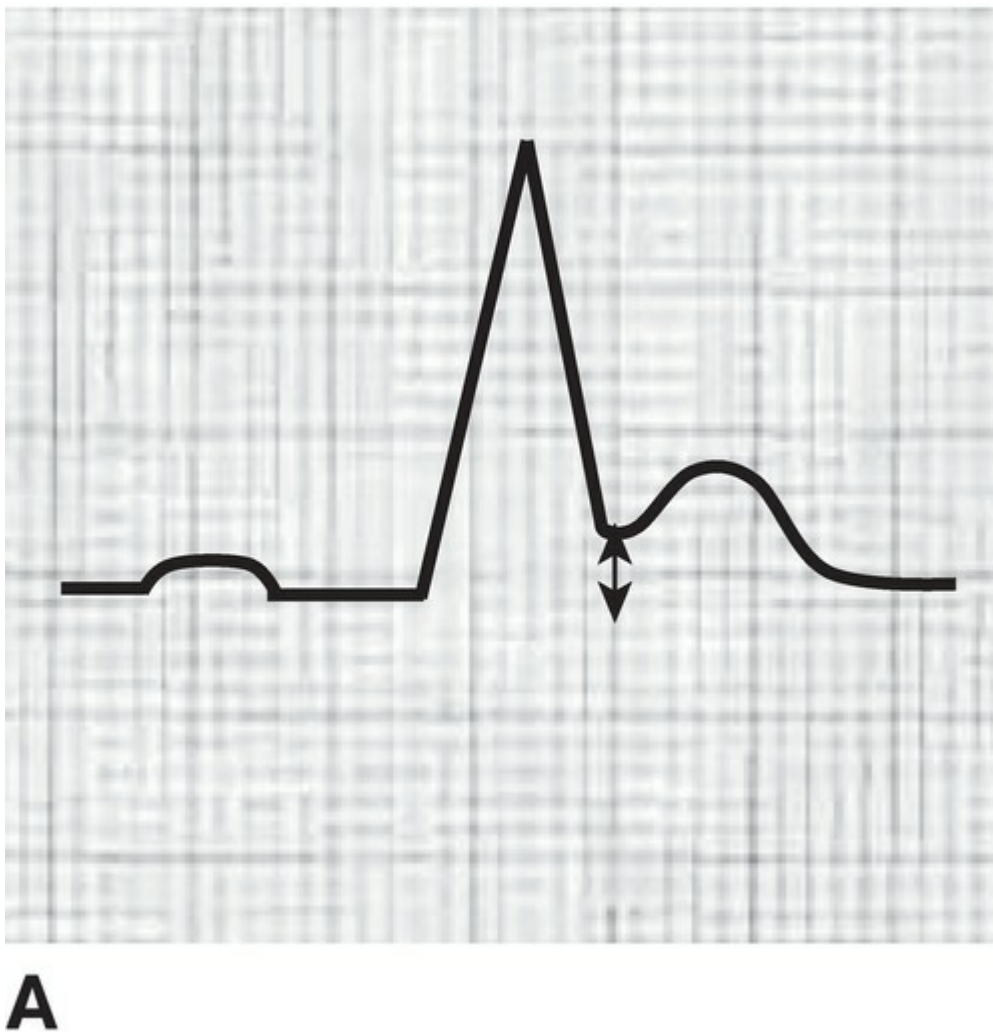


## **B** Anterior Leads

**Figure 71.2** Examples of appropriate discordance.

Published in 2006, the Sgarbossa criteria are a set of validated ECG criteria to help practitioners accurately diagnose an STEMI in the setting of LBBB. These criteria are listed and illustrated in [Figure 71.3](#). A score of 3 or more points is associated with a sensitivity of >97% for the diagnosis of

acute myocardial ischemia. Since the initial publication of the Sgarbossa criteria, multiple studies have shown that the last criterion (Rule 3) has the lowest specificity for acute ischemia. In 2012, Smith and colleagues proposed a modified version of the third Sgarbossa criteria. This modification measures the ratio of the ST segment to the S wave ([Figure 71.4](#)). Acute ischemia is present if this ratio is  $>0.25$ . Both Cai and colleagues and Gregg and colleagues evaluated this modification to the third Sgarbossa criteria and demonstrated increased sensitivity with a specificity that ranges from 90% to 95%.





**B**

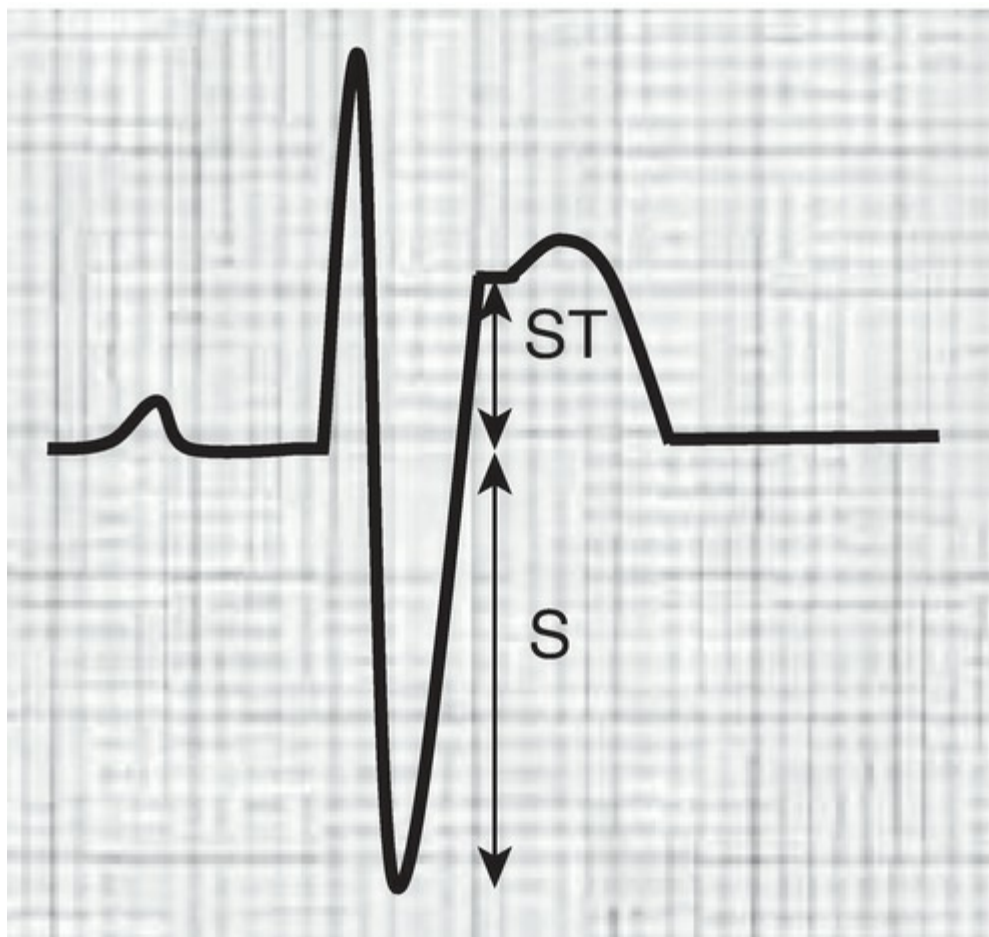




**C**

**Figure 71.3** The Sgarbossa criteria. **Rule 1:** *Concordant* ST-segment elevation  $\geq 1$  mm in any lead (5 points) **(A)**. **Rule 2:** *Concordant* ST-segment depression  $\geq 1$  mm in any one of leads V1 to V3 (3 points) **(B)**. **Rule 3:** *Discordant* ST-segment elevation  $\geq 5$  mm in any lead (2 points) **(C)**.





**Figure 71.4** Smith-modified Sgarbossa criteria. **Rule 3 Modified:** ST-segment elevation  $\geq 25\%$  size of QRS complex ( $ST/S \geq 0.25$ ).

## KEY POINTS

- The Sgarbossa criteria can be used to diagnose STEMI in the setting of an LBBB.
- A Sgarbossa score of 3 or more has a high sensitivity for acute myocardial ischemia.
- The third Sgarbossa criteria, discordance of the ST segment of more than 5 mm, is the least specific for STEMI.
- A modified version of the third Sgarbossa criteria, the ST-segment to S-wave ratio, has a high sensitivity and specificity for acute ischemia.
- Current guidelines recommend reperfusion therapy in patients with an LBBB and:
  - Hemodynamic instability or evidence of acute heart failure
  - A Sgarbossa score of  $\geq 3$
  - A positive modified Sgarbossa criteria
  - Elevated troponin values, regional wall motion abnormalities on

echocardiography that are suggestive of acute myocardial infarction or have evolving ECG changes

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# GETTING AHEAD OF CARDIOGENIC PULMONARY EDEMA: AGGRESSIVE NITROGLYCERIN USAGE

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**SEMHAR Z. TEWELDE, MD**

The management of patients with acute decompensated heart failure (ADHF) has historically focused on the administration of diuretic medications. In fact, the 2013 American Heart Association/American College of Cardiology Foundation Heart Failure Guidelines provide a Class Ia recommendation that “patients with significant fluid overload should initially be treated with loop diuretics given intravenously that should begin in the emergency department without delay.” In recent years, there are increasing data that support the use of high-dose vasodilator therapy as the initial treatment for patients with ADHF. In 2007, the American College of Emergency Physicians published a clinical policy on ADHF, which emphasized the use of vasodilator therapy in the emergency department (ED) management of patients with ADHF.

Importantly, the majority of ED patients who present with ADHF are not volume overloaded. Rather, their pulmonary congestion is due to volume redistribution. The classic ED presentation for these patients includes acute dyspnea, hypertension, and pulmonary edema. Often, these patients arrive to the ED by ambulance and receive sublingual nitroglycerin therapy (0.4 mg every 5 minutes) during their transport. Upon ED arrival, patients should be continued on aggressive vasodilator therapy with nitroglycerin. In most cases, nitroglycerin therapy is started at 50 mcg/min, a dose that is less than what the patient received via the sublingual route with paramedics (80 mcg/min). Numerous studies have demonstrated the importance of initiating a nitroglycerin infusion at a dose of 120 to 200 mcg/min. A report in the *American Journal of Cardiology* noted that at least 120 mcg/min of

nitroglycerin is required to produce a significant decrease in pulmonary capillary wedge pressure. The nitroglycerin infusion can be rapidly increased to 400 mcg/min based upon clinical effect and patient symptoms. Though there have yet to be conclusive data that aggressive vasodilator use improves long-term mortality, it has been shown to prevent intubation and mechanical ventilation in this patient population.

The concomitant use of noninvasive ventilation (NIV) and high-dose vasodilator has been shown to decrease intubation rates, intensive care unit admissions, and hospital length of stay for patients with ADHF. NIV should be initiated early in ED patients with ADHF. Once the patient improves (decreased respiratory rate, decreased dyspnea, improved oxygenation, improved blood pressure), diuretic therapy can be considered. Importantly, diuretics require adequate renal perfusion in order to be effective. During the initial ED evaluation and management when patients are in extremis, renal perfusion is poor, and diuretics are ineffective. Though diuretic therapy is eventually needed in all heart failure patients, they are not beneficial in the initial resuscitation period. High-dose vasodilator therapy and NIV should be the initial tenets of the ED management of patients with AHDF.

## KEY POINTS

- Diuretics should not be considered first-line therapy in the ED treatment of patients with ADHF.
- High-dose nitroglycerin therapy should be initiated early in ADHF, especially in patients who are hypertensive.
- Do not begin a nitroglycerin infusion at a dose less than that provided by the sublingual route.
- Nitroglycerin doses of at least 120 mcg/min are needed to reduce capillary wedge pressure.
- NIV should be administered early and in conjunction with high-dose vasodilator therapy for patients with AHDF.

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# BEYOND DIURESIS: TREATMENT ADJUNCTS IN CARDIOGENIC PULMONARY EDEMA

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NICHOLAS GOODMANSON, MD

Congestive heart failure is the most common reason for hospitalization among U.S. patients over 65 years of age. Acute heart failure can present in a variety of ways that can be conceptually grouped into two pathophysiologic categories: (1) decompensated cardiac failure, the classic accumulation of volume until the patient's chronically depressed pump function is overwhelmed and (2) vascular failure, the rapid increase in afterload through increased systemic vascular resistance leading to increased left ventricular end-diastolic pressure and decreased cardiac output with subsequent pulmonary edema. Diuretics have traditionally been a mainstay of therapy for cardiogenic pulmonary edema. They may not be the preferred initial therapy, however, for those patients with vascular failure, who are often euvolemic, or those with cardiogenic shock, who are often hypovolemic. In these patients, the emergency provider should first optimize preload and afterload reduction with the use of noninvasive positive pressure ventilation (NPPV), nitrates, or inotropes as indicated.

NPPV is an important adjunct in the treatment of patients with cardiogenic pulmonary edema. NPPV decreases preload and afterload by increasing intrathoracic pressure. This decreases venous return (preload) and decreases left ventricular transmural pressure (afterload). NPPV has been shown to reduce the rate of intubation and in-hospital mortality in patients with acute heart failure. Therefore, early use of NPPV in patients with cardiogenic pulmonary edema is warranted in addition to nitrate therapy. Importantly, no studies have demonstrated a difference in patient-centered

outcomes between continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP). An initial setting of 5 to 10 cm H<sub>2</sub>O continuous pressure is recommended for CPAP, while an inspiratory pressure of 10 cm H<sub>2</sub>O and expiratory pressure of 5 cm H<sub>2</sub>O are recommended as initial BiPAP settings.

Nitrates (i.e., nitroglycerin) effectively improve cardiac output for patients presenting with cardiogenic pulmonary edema and hypertension. At low doses, nitrates dilate the venous system and reduce preload. At higher doses, nitrates produce arterial dilation with resultant afterload reduction. This combination decreases myocardial oxygen demand, decreases cardiac work, and ultimately increases cardiac output. Nitroglycerin can be administered first by the sublingual route (with each spray or tablet delivering 400 mcg every 5 minutes) until intravenous access is established and a continuous infusion started. Infusion rates typically begin around 20 mcg per minute, but must be rapidly titrated to clinical effect. In general, the dose can be increased by 40 mcg per minute every 5 minutes to a maximum of 200 mcg per minute. Sodium nitroprusside is an alternative that may be useful in patient's refractory to nitroglycerin therapy. Intravenous doses start at 0.1 mg/kg/min and can be increased by 0.1 mg/kg/min every 5 minutes. Unfortunately, its use is limited by the potential for cyanide toxicity, particularly at high doses and in patients with renal dysfunction.

Though most patients with cardiogenic pulmonary edema present with hypertension, some patients may present with hypotension and cardiogenic shock as the etiology of their edema. Dobutamine is generally the agent of choice in this setting, and an intravenous infusion can be started at 2.5 mcg/kg/min; it can be increased by 2.5 mcg/kg/min to a maximum of 20 mcg/kg/min. It is important to recall that tachydysrhythmias may occur, along with vasodilation and exacerbation of hypotension at higher rates of dobutamine. In this setting, consider starting a vasopressor medication, such as epinephrine or norepinephrine, to augment systemic vascular resistance. Epinephrine, with its increased effect on beta-1 receptors, may be preferred initially over norepinephrine.

Lastly, it is important to avoid the routine use of morphine for patients with cardiogenic pulmonary edema. Though classically recommended for its theoretical ability to reduce afterload, recent retrospective analyses have demonstrated an association between morphine use and increased rates of intubation and in-hospital mortality. Though no large, prospective trials exist, the routine use of morphine in patients with cardiogenic pulmonary edema should be avoided.

As with most patients presenting to the emergency department, diagnosis

and management must occur simultaneously. It is helpful to consider two categories of cardiogenic pulmonary edema: decompensated cardiac failure and vascular failure. If true volume overload is suspected, diuretics and ACE inhibitors can then be added after the administration of nitrate therapy and NPPV.

## KEY POINTS

- Early use of NPPV significantly reduces rates of intubation and in-hospital mortality.
- Nitrates should be administered early and rapidly to maximize preload and afterload reduction.
- For patients presenting with pulmonary edema secondary to cardiogenic shock, an inotrope such as dobutamine should be initiated to improve cardiac output.
- Recent data suggest worse outcomes with the use of morphine for patients with cardiogenic pulmonary edema.
- Diuretic administration should follow the use of more rapidly acting preload- and afterload-reducing therapies, particularly if there is little suspicion of volume afterload.

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# KNOW HOW TO DIFFERENTIATE CARDIAC VERSUS NONCARDIAC CAUSES OF SYNCOPE

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**OMOYEMI ADEBAYO, MD**

Syncope is defined as a transient loss of consciousness, characterized by the absence of postural tone, and is caused by global hypoperfusion to the brain. Importantly, patients with syncope rapidly return to their baseline mental status. When assessing the patient with syncope, it is critical to differentiate between cardiac and noncardiac etiologies. Patients with a cardiac cause of syncope have markedly higher morbidity and mortality compared with patients who have a noncardiac etiology. The history of present illness (HPI), physical examination, electrocardiogram (ECG), and directed diagnostic testing can provide valuable information in identifying a cardiac cause of syncope.

The HPI, including the patient's past medical history and family history, is essential in the evaluation of syncope and provides valuable clues to a cardiac etiology. A past history of congestive heart failure, valvular heart disease, coronary artery disease, or other structural cardiac abnormalities increases the likelihood of a cardiac cause for syncope. These factors are included in numerous syncope scoring systems and clinical decision rules. In younger patients without pre-existing cardiac disease, it is still critical to consider a cardiac etiology of syncope. Prodromal symptoms such as lightheadedness, palpitations, shortness of breath, and chest pain may be more common in patients with a cardiac cause for syncope. In addition, patients who report syncope when in a supine position or during exertion should be evaluated for a cardiac etiology. Signs and symptoms that suggest a seizure, such as myoclonus or incontinence, can be seen in patients with

syncope and have been reported with cardiac causes of syncope. Notwithstanding, the absence of specific symptoms should not be used in isolation to exclude a cardiac cause of syncope. A family history of premature cardiac disease or sudden, unexplained death is important to note.

In addition to the HPI, the physical examination is critical and should be directed toward identifying abnormal findings that suggest a cardiac, or noncardiac, cause of syncope. It is common to obtain orthostatic vital signs in patients with syncope. Knopp and colleagues demonstrated that an increase of 30 beats or more in heart rate associated with lightheadedness upon standing was more sensitive and specific at identifying orthostatic hypotension compared with changes in blood pressure readings. While positive orthostatic vital signs suggest a noncardiac cause of syncope, it is important to recognize the limitations of these measurements. Numerous studies have demonstrated the inaccuracy of orthostatic vital sign measurements in older patients. Positive, or negative, orthostatic measurements cannot completely exclude a cardiac cause of syncope. Additional physical examination findings that indicate cardiac abnormalities and increase the likelihood of a cardiac cause for syncope include a murmur, irregular rhythm, or signs of congestive heart failure.

As aforementioned, the ECG is a fundamental component in the evaluation of a patient with syncope. The ECG should be scrutinized for signs of arrhythmias, ischemia, and structural disorders. Important diagnoses to consider that produce characteristic ECG findings are listed in [Table 74.1](#).

**TABLE 74.1 ABNORMAL ECG FINDINGS OF CARDIAC SYNCOPE**

| CHANNELOPATHIES  | VASCULAR ABNORMALITIES   | ELECTROLYTE ABNORMALITIES   | STRUCTURAL ABNORMALITIES  | CONDUCTION ABNORMALITIES   |
|--|--|---|---|--|
| <ul style="list-style-type: none"> <li>■ Brugada</li> <li>■ Long QT syndrome</li> <li>■ Short QT syndrome</li> </ul> | <ul style="list-style-type: none"> <li>■ ACS</li> <li>■ PE</li> <li>■ Cardiac tamponade</li> </ul> | <ul style="list-style-type: none"> <li>■ Hyper K<sup>+</sup></li> <li>■ Hypo K<sup>+</sup></li> <li>■ Hypo Ca<sup>2+</sup></li> </ul> | <ul style="list-style-type: none"> <li>■ HOCM</li> <li>■ WPW</li> </ul> | <ul style="list-style-type: none"> <li>■ AV Blocks</li> <li>■ SVT</li> </ul> |

ACS, Acute coronary syndrome; PE, Pulmonary embolism; K<sup>+</sup>, Potassium; Ca<sup>2+</sup>, Calcium; HOCM, Hypertrophic cardiomyopathy; WPW, Wolff-Parkinson-White; AV, Atrioventricular; SVT, Supraventricular tachycardia.

Routine laboratory testing is seldom useful in the evaluation of syncope. Cardiac laboratory studies (e.g., troponin, B-type natriuretic peptide, and D-

dimer) have been studied to assess sensitivity and specificity in identifying a cardiac cause of syncope. Results from this limited literature have been indeterminate, and their routine use cannot be recommended.

When the etiology of syncope is uncertain despite a thorough history, physical examination, and ECG, scoring systems and decision rules can be used to risk stratify patients. Common decision rules include the OESIL Scoring System and San Francisco Syncope Rules. At present, studies on the use of select scoring systems and decision rules have produced inconsistent results. While helpful, they have not been shown to be superior to clinical gestalt.

## KEY POINTS

- A past medical history of congestive heart failure, valvular abnormalities, or coronary artery disease increases the likelihood of a cardiac etiology of syncope.
- Syncope that occurs with exertion or in a supine position suggests a cardiac etiology.
- Orthostatic vital sign measurements are unreliable in the elderly patient.
- The ECG is an essential tool in assessing cardiac causes of syncope.
- Risk stratification scores (OESIL, San Francisco Syncope Rule) can be utilized in the evaluation of syncope patients but remain controversial.

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## PEARLS IN SYNCOPE ECG INTERPRETATION

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**CARMEN AVENDANO, MD AND SEMHAR Z. TEWELDE,  
MD**

Syncope is defined as a loss of consciousness that results from insufficient blood flow to the brain. Syncope accounts for more than 700,000 emergency department (ED) visits and 6% of hospital admissions each year in the United States. Though most etiologies are benign, a small portion of patients have a life-threatening cause of their syncope. The history of present illness, physical examination, and 12-lead electrocardiogram (ECG) are fundamental to the ED evaluation of a patient with syncope. In fact, it is often the ECG that provides critical clues to the etiology of potentially life-threatening causes of syncope. In the ED patient with syncope, the ECG should be scrutinized for signs of ischemia, bradydysrhythmias, tachydysrhythmias, and conduction delays. Additional critical diagnoses to consider that can be detected with the ECG include ventricular preexcitation syndromes, Brugada syndrome, prolonged or short QT syndromes, hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, and catecholaminergic polymorphic ventricular tachycardia. The ECG characteristics and pearls for these life-threatening causes of syncope include the following:

### **VENTRICULAR PREEXCITATION (E.G., WOLFF-PARKINSON-WHITE SYNDROME)**

- PR segment < 120 ms
- QRS complex > 110 ms
- Slurred upstroke of the initial part of the R wave (delta wave)

- Type A: left-sided accessory pathway—delta wave in all precordial leads,  $R > S$  in lead V1
- Type B: right-sided accessory pathway—negative delta waves in leads V1 and V2

## **BRUGADA SYNDROME**

- Type 1
  - Coved ST-segment elevation
  - $>2$  mm of ST-segment elevation in 2 or more precordial leads (V1 to V3) followed by negative T wave
- Type 2
  - Saddle back ST-segment elevation
  - $>2$  mm of ST-segment elevation in 2 or more precordial leads (V1 to V3)
- Type 3
  - Either type 1 or 2 with  $<2$  mm ST-segment elevation

## **PROLONGED QT SYNDROME**

- $QTc = QT/\sqrt{R-R \text{ interval}}$
- Prolonged if  $QTc > 440$  ms in men or  $>460$  ms in females
- Increased risk of dysrhythmias when the  $QTc > 500$  ms
- Potential for a “R on T” phenomenon, where a premature ventricular contraction at the end of T wave can induce polymorphic ventricular tachycardia or torsades de Pointes.
- Etiologies to consider include electrolyte deficiencies (potassium, magnesium, calcium), hypothermia, cardiac ischemia, increased intracranial pressure, and toxins.

## **SHORT QT SYNDROME**

- Shortened if  $QTc < 330$  ms in males or  $<340$  ms in females
- Short, or absent, ST-segment with peaked appearance of the T wave
- Etiologies to consider include congenital shortening, digoxin toxicity, and hypercalcemia.

## **HYPERTROPHIC CARDIOMYOPATHY (HOCM)**

- Deep Q waves in the lateral (I, aVL, V5-V6) and inferior (II, III, aVF) leads
- Left ventricular hypertrophy (LVH)
- Left atrial enlargement
- Apical HCOM variant
  - Localized hypertrophy of the LV apex
  - LVH
  - Giant T-wave inversions in the precordial leads
  - Possibly inverted T waves in the inferior and lateral leads

## **ARRHYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA (ARVD)**

- Epsilon wave (small positive deflection at end of QRS complex) is the most specific finding and found in ~30% of patients.
- T-wave inversions in leads V1 to V3
- Prolonged QRS in leads V1 to V3 (100 to 120 ms)
- Slurred S-wave upstroke in leads V1 to V3 (50 to 55 ms)
- Consider ARVD in patients with paroxysmal episodes of ventricular tachycardia.

## **CATECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA (CPVT)**

- ECG is typically normal at rest.
- Exercise classically induces ventricular tachycardia from adrenergic activation.
- Bidirectional tachycardia (alternating 180 degrees QRS axis beat to beat variation)
- Ventricular ectopy is often noted when the heart rate increases above 100 beats per minute.

### **KEY POINTS**

- An ECG should be performed on all ED patients with syncope.
- Scrutinize the ECG for signs of ischemia, conduction delay, tachydysrhythmias, and bradydysrhythmias.

- Be sure to look for signs of ventricular pre-excitation syndromes, Brugada syndrome, prolonged or short QT syndrome, ARVD, HOCM, and CPVT.
- Consider electrolyte abnormalities in the patient with QT interval abnormalities.
- The epsilon wave is the most specific finding for ARVD.

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# SYNCOPE: AVOIDING A SHOTGUN WEDDING

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**OMOYEMI ADEBAYO, MD**

Syncope represents up to 5% of annual emergency department (ED) visits in the United States. In the evaluation of ED patients with syncope, the emergency provider (EP) should attempt to answer the following two questions: (1) what caused the event and (2) what is the patient's risk for adverse outcome as a result of this event? The ability to answer these two questions is predicated on the history of present illness (HPI), physical examination, electrocardiogram (ECG), and select diagnostic testing.

Syncope is described as a transient loss of consciousness caused by a period of cerebral hypoperfusion and is characterized by the inability to maintain postural tone followed by a rapid recovery. Deadly etiologies of syncope to consider include subarachnoid hemorrhage (SAH), ruptured abdominal aortic aneurysm, aortic dissection, pulmonary embolism (PE), ruptured ectopic pregnancy, and acute coronary syndrome. Approximately 5% to 15% of patients with these conditions may present with syncope. During the HPI, the EP should ask about preceding symptoms that suggest a life-threatening etiology of syncope, such as headache, abdominal pain, back pain, chest pain, or shortness of breath. It is sometimes difficult to differentiate seizure and stroke from syncope, especially when reliable sources for the HPI are lacking. In fact, patients with syncope can have myoclonus or urinary incontinence, symptoms classically attributed to seizure activity. In patients in whom a targeted HPI and physical exam fail to determine the cause of syncope, it is tempting to order an array of laboratory or diagnostic imaging studies to further risk stratify patients. This should be avoided.

An ECG provides a diagnosis in ~5% of cases and should be obtained in



all patients who present with syncope. The ECG should be reviewed for signs of ischemia or arrhythmia. It is also important to review the ECG for signs of preexcitation syndromes, Brugada syndrome, hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, and prolongation of the QT interval. ECG findings specific for these conditions are discussed in other chapters in this textbook.

Though hypoglycemia is an exceedingly rare cause of syncope, a blood glucose value should be obtained and included with the initial vital sign measurements. Orthostatic vital sign measurements may be helpful in young patients who do not have significant comorbid conditions. However, it is important to recognize the limitations of orthostatic measurements. Numerous studies have demonstrated the inaccuracy of orthostatic vital signs in older patients, especially those taking beta-blocker or calcium-channel blocker medications. Do not rely on orthostatic measurements in the elderly.

Routine laboratory testing or diagnostic imaging is seldom useful in the patient with syncope, unless suggested by findings from the HPI and physical examination. Cardiac laboratory studies (troponin, D-dimer) have not been shown to reliably differentiate patients with a cardiac cause from those who have a noncardiac etiology of syncope. A complete blood count rarely detects a critical anemia that is not suggested by the physical examination. Similarly, a comprehensive metabolic panel rarely yields useful information about electrolyte disorders that are not suggested by abnormalities on the ECG. Computed tomography (CT) of the brain is useful when the diagnosis of SAH is suspected. Notwithstanding, evidence to support the use of a CT of the head in all patients with syncope patient is lacking. Transthoracic echocardiography should be considered in patients with a suspected cardiac structural abnormality, such as an atrial myxoma, ventricular septal thickening, or valvular disorder. In many cases, the EP can appropriately risk stratify patients without the need for laboratory testing or advanced diagnostic imaging.

A number of tools have been developed and validated to assist the EP in the risk stratification of patients with syncope. These tools use different combinations of elements of the HPI, patient comorbidities, ECG findings, and laboratory tests. While these syncope decision rules do help to identify which patients are at high risk of adverse outcome, they have not been shown to be better than physician gestalt.

## KEY POINTS

- Deadly causes of syncope include SAH, ruptured abdominal aortic aneurysm, aortic dissection, PE, ruptured ectopic pregnancy, and acute coronary syndrome.
- The HPI, physical examination, and ECG are essential in the evaluation of syncope.
- Myoclonus and urinary incontinence can be seen in patients with syncope.
- Hypoglycemia is a rare cause of syncope.
- Laboratory testing and diagnostic imaging should be ordered when suggested by findings from the HPI, exam, and ECG.

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## **SECTION IV**

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# **GASTROENTEROLOGY**

# WHEN AN APPY DOESN'T FOLLOW THE RULES

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**CAROLINE BRANDON, MD**

Acute appendicitis is the most common abdominal emergency. It can occur at any age, but most commonly presents during the second decade of life. Appendicitis has a male predominance, with a lifetime risk of 9% in males and 6% in females. It occurs when there is obstruction of the appendiceal lumen, from a fecalith, tumor, infection, or hyperplasia of lymphoid follicles.

Classic symptoms of appendicitis include pain that is initially vague and periumbilical followed by a more localized parietal pain in the right lower quadrant. Only 50% of people present with these classic symptoms; in the other 50%, appendicitis can be difficult to diagnosis.

Appendicitis pain does not always occur in the right lower quadrant. Patients with congenital abnormalities may present with left-sided abdominal pain. While most patients with midgut malrotation or situs inversus totalis are diagnosed in the first month of life, some patients are not diagnosed until they present with acute appendicitis. Retrocecal appendicitis may cause right upper quadrant pain. Retrocecal appendices are found in 26% to 65% of the population, leading to a different pattern of inflammation. Appendiceal abscesses may develop in the pararenal space, retrocolic and paracolic gutters, and even in the subhepatic area. Rarely, patients with acute appendicitis may present with chest pain or epigastric pain. Patients with undiagnosed congenital diaphragmatic hernia may have an inflamed appendix in the chest cavity, which would present as chest pain.

Importantly, there are three specific patient populations who are more likely to present with atypical pain from appendicitis: pregnant women, children <5 years of age, and elderly patients.

Acute appendicitis is the most common nonobstetric abdominal emergency during pregnancy. Patients may endorse burning epigastric pain, changes in bowel habits, rectal and/or vaginal pain. Nausea, vomiting, and nonspecific abdominal pain may be attributed to morning sickness in the first trimester and lead to a delayed diagnosis. If patients are in the third trimester, the appendix is located higher up in the abdomen, and patients may complain of right flank or right upper quadrant pain. Ultrasound (US) is 67% to 100% sensitive for appendicitis in pregnant patients if the appendix is visualized. When it is not visualized, magnetic resonance imaging (MRI) is recommended. The risk to the fetus increases if there is a delay to diagnosis and the appendix perforates (fetal loss is 20% to 30% with perforation vs. <5% without).

Young children with appendicitis present differently than adults. Infants may have only lethargy and abdominal distention, while toddlers usually present with vomiting and diarrhea, which may be attributed to a viral syndrome. Over two-thirds (70%) of children <3 years old will perforate within 48 hours due to delayed diagnosis. Moreover, two-thirds (67%) of acute appendicitis cases in young children are not diagnosed on the initial visit to a provider. Repeat abdominal examinations will aid in diagnosis, and strong, clear return precautions for worsening pain, focal pain, or lack of resolution of symptoms are extremely important.

If there is concern for an acute appendicitis in a child or pregnant woman, US followed by MRI (if US nondiagnostic) is recommended. When the appendix is seen on MRI, there is close to a 100% sensitivity and specificity for the diagnosis of acute appendicitis. If MRI is not available, surgical consultation should nonetheless be obtained when history, examination, and laboratory studies are suspicious for acute appendicitis. Computed tomography (CT) with its attendant risk of ionizing radiation may be an option for definitive diagnosis in both pregnancy and children, although risks and benefits should be discussed with the patient, parents, and specialty consults.

Appendicitis accounts for 7% of abdominal pain in elderly patients presenting to the emergency department. These patients often have multiple comorbid conditions that obscure the diagnosis. Less than half of the patients over 60 years of age present with typical migratory abdominal pain; instead, they have vague or generalized abdominal pain. They also present to the emergency department (ED) later in the disease course, leading to higher rates of perforation. Studies indicate that the majority of elderly patients with acute appendicitis will have already perforated by the time of surgery. This results in a very high risk of morbidity and mortality when elderly patients develop appendicitis. CT is widely considered first line for evaluating elderly

patients for appendicitis. In this population, there are a number of other potentially life-threatening diagnoses that may be detected on CT, and the long-term risks of radiation are of less concern.

## KEY POINTS

- Only 50% of appendicitis cases will present with the classic periumbilical pain migrating to the right lower quadrant.
- Patients with congenital abnormalities or retrocolic and paracolic gutters may present with right upper, left-sided, or even chest pain.
- Pregnant women, children under the age of 5, and the elderly are specific populations in whom to expect atypical presentations of appendicitis.

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# **ANALGESIA FOR THE PATIENT WITH ACUTE ABDOMINAL PAIN: DON'T DELAY!**

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**ADRIAN FLORES, MD, MPH**

Should a patient with acute abdominal pain be given pain medication prior to evaluation by a surgeon? In an emergency department (ED) setting, the simple answer is yes.

An acute abdomen is severe abdominal pain of generally <24 hours duration, caused by potentially life-threatening intra-abdominal pathology that requires prompt attention, accurate diagnosis, and often surgical intervention. Surgical tradition discouraged the use of pain medications, specifically narcotics, fearing that it might mask clinical symptoms and lead to a delay in diagnosis. At one time, this may have been true. It is thought to have originated a century ago when patients were given very high doses of morphine rendering them unresponsive.

*Cope's Early Diagnosis of the Acute Abdomen*, a seminal surgical text first published 89 years ago, emphasized and reinforced this message until the 1987 edition, when its stance began to soften slightly. Not until the 22nd edition in 2010 did the message change, describing the withholding of pain medication as a “cruel practice that should be condemned” while acknowledging that “it will take several generations of physicians to eliminate (this belief because) the rule has become so firmly ingrained in the minds of physicians.”

Policy statements by the Joint Commission on Accreditation of Healthcare Organizations (JCAHO), the Agency for Health Care Policy and Research (AHCPR), and the American College of Emergency Physicians

(ACEP) cite evidence that pain control does not mask clinical findings or delay diagnosis. Nevertheless, pain continues to be undertreated in emergency and inpatient settings. Some physicians even still opt to withhold medications until evaluation by a surgeon.

So it appears clear that we should provide pain relief. How do we go about it? What medications should we give and what dose? In acute abdominal pain, intravenous (IV) medications are preferred. They are easier to titrate and will help keep the patient NPO (nothing by mouth) in anticipation of surgery. Using morphine as a reference point, the starting therapeutic dose is 0.1 mg/kg IV, keeping in mind that some opiate naive patients are very sensitive while other patients may have a high tolerance from chronic use. Though the starting dose of morphine is usually 7 to 8 mg (based on a 70- to 80-kg patient), it is appropriate to give patients  $\frac{1}{2}$  to  $\frac{3}{4}$  of a dose with PRN (as needed) instructions for the nurse to repeat the dose in 15 to 20 minutes if the initial dose is ineffective. The availability of a repeat dose in the hands of a responsible and trusted nurse is important to ensure that your patient's pain is being treated in a timely matter, especially in a busy ED. Frequent reassessment, when possible, also helps to attain this goal. Also, consider a higher starting dose based on equivalents of home doses in patients that take chronic pain medication.

An opioid equivalency table (available on most smartphone pharmacopeias) is important to help determine the equivalent dose of IV for a PO (oral) medication that the patient chronically takes or to switch between classes of IV opioids. For instance, 10 mg of IV morphine is equivalent to 30 mg of PO morphine, which is equivalent to 1.5 mg of IV hydromorphone. However, even if a patient did not respond to, or is tolerant to, 10 mg of IV morphine, there is incomplete cross-tolerance when switching between opioids. A smaller dose of hydromorphone may be effective in controlling pain, so consider decreasing the initial dose rather than giving exact equivalent.

While morphine is the reference standard for dosing opioids, there may be better alternatives in the appropriate clinical setting. Morphine is often poorly tolerated, causing nausea and pruritus from histamine release. Hydromorphone tends to cause these side effects less often. Both medications can cause hypotension, so consider fentanyl (less hemodynamic effects) for pain control in an unstable patient. But remember, fentanyl is shorter acting (45 minutes), and good pain control results from a steady basal rate of pain medication. Switch to a longer-acting IV opioid when appropriate to avoid leaving your patient in severe pain between doses of short-acting medications. Even though oral opiate formulations are longer acting than IV opiates, avoid the use of oral pain medication for acute



abdominal pain. Oral opiates are harder to titrate because of the longer and less predictable time to onset. Once you establish that your patient does not have a surgical diagnosis, you can consider a longer-acting oral regimen.

## KEY POINTS

- Do not withhold opioid analgesic pain medications for patients presenting with acute abdominal pain.
- Pain control does not mask clinical findings or lead to a delay in diagnosis.
- Always consider patient tolerance, duration of action, and timely redosing to achieve the goal of adequate pain control.

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## GET TO IT EARLY: SIGMOID VOLVULUS

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**JORGE ONTIVEROS, MD**

Sigmoid volvulus occurs when a portion of the sigmoid colon twists around its mesentery causing obstruction of the intestinal lumen. It typically requires a redundant sigmoid colon attached to a narrow mesentery to occur. Obstruction of the lumen occurs when the degree of torsion exceeds 180 degrees, and compromise of the vascular supply occurs after torsion exceeds 360 degrees.

Sigmoid volvulus may be an acquired or congenital condition. In North America, sigmoid volvulus is rare, accounting for only 3% to 5% of all intestinal obstructions. In contrast, sigmoid volvulus accounts for 20% to 54% of all intestinal obstructions in high incidence areas (Latin America, Africa, Middle East, Eastern Europe). The higher incidence of sigmoid volvulus in those areas is thought to be secondary to higher rates of redundant sigmoid colon, as seen in anatomic studies. Furthermore, Chagas disease is frequently associated with sigmoid volvulus. In low incidence areas, sigmoid volvulus is associated with constipation, which is believed to increase risk of volvulus via elongation and dilation of the colon. Other risk factors include older age, diabetes, neuropsychiatric diseases, high-fiber diet, and residing in long-term care facilities. Sigmoid volvulus is exceedingly rare in children. Nonetheless, chronic constipation and underlying comorbidities (Hirschsprung disease, intestinal malrotation, mental retardation) are risk factors.

Sigmoid volvulus classically presents with abdominal pain, distention, and constipation. The diagnosis may be relatively close at hand in patients with acute obstruction: sudden onset of severe pain with gross abdominal distention. However, the presentation may be insidious with mild and slowly

progressive abdominal pain and constipation. Patients may also present with diarrhea if there is incomplete obstruction of the intestinal lumen. Furthermore, sigmoid colon torsion and spontaneous detorsion may occur, leading patients to report prior episodes of similar symptoms. Late presentation is common: the majority of patients present 3 to 4 days after the start of symptoms while only 17% present within 48 hours of onset.

Barium enema, computed tomography (CT), and magnetic resonance (MR) have all been used to diagnose sigmoid volvulus. However, simple plain abdominal films may be diagnostic in up to 80% of sigmoid volvulus cases. Images will reveal a dilated sigmoid colon: a large area of hyperlucency extending from the pelvis, resembling a coffee bean or bent inner tube. Air-fluid levels and the absence of rectal gas may also be noted. In equivocal cases, contrast enema or CT can be used to make the diagnosis. Contrast enema will give the appearance of a bird's beak at the location of torsion. On CT, the whirl sign (evidence of structures twisting on themselves, mimicking a whirlpool) and dilated sigmoid colon may be seen. Delay in diagnosis can lead to compromise of vascular perfusion causing bowel ischemia, gangrene, and perforation.

Treatment of sigmoid volvulus depends on patient characteristics, comorbidities, and presentation. If there is any evidence of peritonitis, perforation or bowel gangrene, immediate surgical consultation for laparotomy is indicated. Surgical treatments include detorsion, mesopexy, resection and primary anastomosis, or resection and stoma. In the absence of peritonitis or gangrene, both gastroenterology and surgery should be consulted early. Sigmoidoscopy/colonoscopy can be diagnostic and therapeutic with endoscopic decompression and detorsion, but surgery is indicated if the bowel is noted to be gangrenous during endoscopy or if detorsion is unsuccessful. Detorsion with colonoscopy has been reported to be successful 70% to 90% of the time. However, retorsion after decompression is common, thus elective surgical treatment even after successful detorsion is recommended. Mortality associated with sigmoid volvulus exceeds 50% in patients with gangrene while mortality in patients without gangrene is <10%. Increased mortality is associated with delay in detorsion, shock, gangrene, perforation, leukocytosis, electrolyte abnormalities, and advanced age.

## KEY POINTS

- Patients with sigmoid volvulus may present with subtle and even

paradoxical symptoms (vague abdominal pain, mild abdominal distention, constipation, or diarrhea).

- Risk factors include older age (mean age 60 to 70), institutionalization, neuropsychiatric diseases, and chronic constipation.
- Plain films are high yield and usually diagnostic.
- Once the diagnosis is made, consult gastroenterology and surgery! Endoscopic decompression and detorsion is often successful and must be performed before gangrene sets in.

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# **CECAL VOLVULUS: DON'T MISS IT!**

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**JAN MARIE SHOENBERGER, MD**

Abdominal pain is a common emergency department (ED) presentation with a variable differential diagnosis depending on several important factors such as age, gender, and clinical history. Because the common etiologies are the ones we see most often and are most familiar with, we often forget to consider the more unusual causes of acute abdominal pain. Cecal volvulus is definitely on that list as it is infrequently encountered. A recent study looking at patients with bowel obstruction admitted to hospitals in the United States between 2002 and 2010 found that colonic volvulus (including both cecal and sigmoid volvulus) was found to be the cause of the bowel obstruction in <2% of cases. Though rare, the authors of the same study found that the mortality rate for cases of cecal volvulus was almost 7%.

Cecal volvulus can present at any age and is a result of anatomic abnormalities that occur during intestinal development in embryogenesis. There is also a relationship with previous abdominal surgery that is theorized to involve postoperative adhesions that later become points of fixation allowing volvulus to occur. Other conditions such as chronic constipation, distal colon obstruction, ileus, late-term pregnancy, and high fiber intake have also been linked to cecal volvulus. Additionally, concurrent illness or hospitalization leads to an increased propensity for cecal volvulus possibly due to an increased occurrence of colon distention and intestinal dysmotility.

There are three patterns of clinical presentation of cecal volvulus that have been described. The first is a recurrent, intermittent pattern that occurs in about half of patients before they are ultimately diagnosed with cecal volvulus. Patients have recurrent episodes of right lower quadrant pain, abdominal distention, and relief of symptoms after flatus. The second type of

presentation is that of acute obstruction from the volvulus. This presentation is clinically indistinguishable from acute small bowel obstruction and would likely be specifically identified on imaging prior to surgery or intraoperatively. The third type of presentation is a fulminant one that develops if acute volvulus is not initially recognized and the patient develops intestinal strangulation and perforation.

The patient's history and physical examination will most likely lead the clinician to be suspicious of intestinal obstruction, either acute or intermittent. Symptoms such as vomiting, crampy abdominal pain, and abdominal distention may be present. No laboratory values will confirm the diagnosis; it will either be made by imaging or intraoperatively if the patient is deemed an emergent surgical candidate.

Imaging options include an abdominal x-ray (plain film) series or advanced imaging such as computed tomography (CT). Plain films will show signs consistent with intestinal obstruction, such as air-fluid levels and dilated loops of bowel. They may show the classic "coffee-bean" sign, which is a view of the dilated, twisted cecum with air and fluid pointing to the left upper quadrant. Another possible x-ray sign is the "bird beak" sign that reflects progressively tapering efferent and afferent loops of bowel terminating at the site of the torsion. More likely than not, however, the abdominal x-rays will be abnormal in some way but will not be specific enough to make the definitive diagnosis of cecal volvulus. In these cases, a CT is indicated.

Surgical management is the preferred treatment for cecal volvulus with the goal of correcting the intestinal obstruction. Resection of the affected segment of colon is occasionally required in cases where necrosis has already occurred, and some surgeons favor this approach to prevent recurrent volvulus.

## KEY POINTS

- Patients with cecal volvulus may present with classic signs of obstruction, or intermittent crampy right lower quadrant pain with abdominal distention.
- Abdominal x-rays may show a "coffee-bean" or "bird beak" sign, but if they are nondiagnostic, CT should be performed if the diagnosis is suspected.
- The management is surgical.

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## **ALTERED MENTAL STATUS IN A CHILD: DON'T FORGET ABOUT INTUSSUSCEPTION!**

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**AARON E. KORNBLITH, MD AND JEFFREY BULLARD-BERENT, MD**

Intussusception occurs when a proximal segment of bowel telescopes into the more distal segment. The most frequent location is between the ileum and colon (ileocolic intussusception). This leads to venous and lymphatic congestion with resulting intestinal edema. Left untreated, intussusception leads to arterial compromise, intestinal infarction, peritonitis, and even perforation.

In infants and young children, intussusception is the most common cause of bowel obstruction. Most cases occur between the ages of 5 months and 5 years, and over 80% occur in children <2 years of age. These young patients are at increased risk for delayed diagnoses because of their poor ability to communicate. Ileocolic intussusception is more common in boys and is characterized by seasonal variation, corresponding to peaks in viral gastroenteritis. The classic finding of “currant jelly” stool is present in <50% of cases, and the combination of bloody stools, abdominal pain, vomiting, and abdominal mass is found together in <20%.

It wasn't until 1979 that the medical literature recognized intussusception as a cause of isolated altered mental status (AMS) in children, and the pathophysiology of AMS from intussusception is still unclear. Children with AMS from intussusception have been characterized as being listless, weak, sleepy, comatose, apathetic, or even lifeless. These symptoms make it easy for emergency physicians to overlook intussusception until other more



readily recognized causes of AMS are excluded or until more classic symptoms develop. AMS in the setting of intussusception does not portend more severe disease, but it may delay the diagnosis. A delay in diagnosis decreases likelihood of successful reduction of intussusception by air or liquid enema such that surgical reduction may be required.

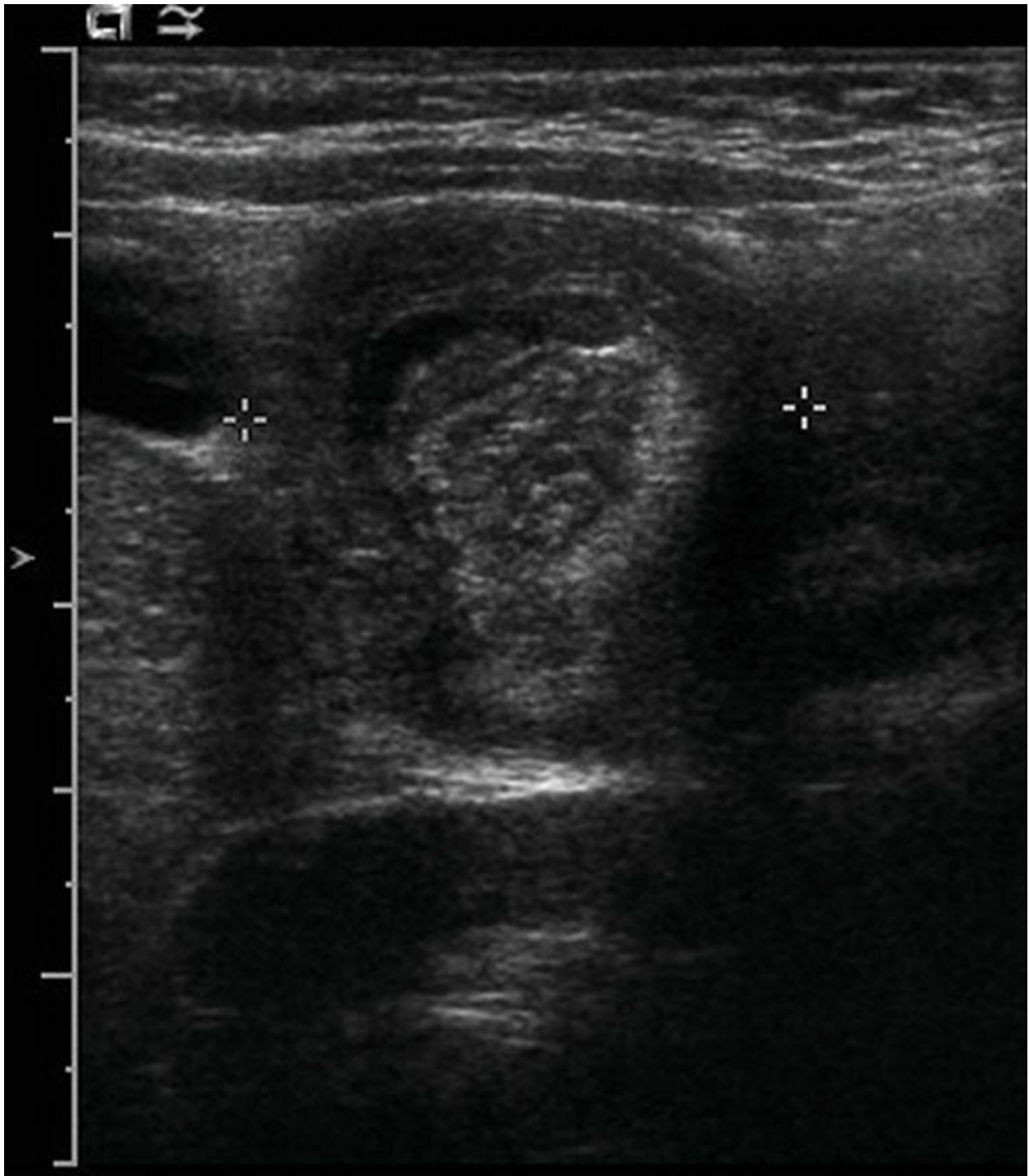
How then is it possible to screen a child with unexplained AMS for intussusception? The gold standard of diagnosis is air or liquid contrast enema, but radiology-performed ultrasound is equally sensitive and specific in the hands of an experienced ultrasonographer and has become first line in the diagnosis. However, both require expertise and a trip away from the resuscitation area. In contrast, three diagnostic tests can be performed quickly at the bedside to alter the posttest probability of intussusception when other diagnostic studies are not available: fecal occult blood testing (FOBT), point of care (POC) ultrasound, and abdominal radiography.

Fecal occult blood is an important predictor of intussusception. As the intestinal lining begins to slough, it will eventually show the classic currant jelly stools. FOBT may be positive much earlier. In a retrospective analysis, stool with occult blood was significantly more common in patients with intussusception than in those without blood. Moreover, among patients with intussusception whose history or examination indicated no gross rectal bleeding, occult blood was found in 75%. Though FOBT is far from foolproof, a positive result is associated with intussusception and therefore increases the likelihood of the diagnosis.

POC ultrasound is a test that can help rule in intussusception. In one prospective study, emergency providers with only an hour of training proved quite adept at diagnosing intussusception using POC ultrasound. The classic ultrasound image of intussusception is a “target sign” representing layers of the intestine within intestine ([Figure 81.1](#)), seen most often in the right upper quadrant. Seeing the intussusception is clearly helpful, but POC ultrasound is operator dependent and not sensitive enough to rule out the diagnosis. If concern for intussusception is present and target sign not seen on POC ultrasound, further imaging should be obtained.



**A**



**B**

**Figure 81.1** Ultrasound image of a “target sign” representing an ileocolic intussusception. Transverse views of intussusception demonstrate a classic concentric ring sign **(A)** and characteristic hyperechoic, heterogenous intussusceptum surrounded by edematous bowel wall of the containing intussuscipiens **(B)**. From Cosby KS, Kendall JL, eds. *Practical Guide to Emergency Ultrasound*. 2nd ed. Philadelphia, PA: Wolters Kluwer Health, 2013.

Plain abdominal radiographs are not sensitive for diagnosing intussusception, but certain findings decrease a patient's likelihood of having it. In intussusception, air in the ascending colon is replaced by small bowel telescoping inside the lumen, so one would see an *absence* of bowel gas in the ascending colon and right upper quadrant. In a prospective study of children aged 3 months to 3 years who were suspected of having intussusception, when air was present in the ascending colon in *all three* views (supine, prone, and left lateral decubitus), the sensitivity for excluding intussusception was 100% (95% confidence interval, 79.1 to 100). However, the ability to exclude intussusception drops off if air is seen in the ascending colon in only one or two views. Nevertheless, abdominal plain films can be performed at the bedside in an unstable patient.

## TREATMENT

Patients with hemodynamic instability or abnormal vital signs should be resuscitated with intravenous fluids. Evidence of perforation or peritonitis mandates a surgical consult. In a stable patient, air, water, or barium enema with fluoroscopic or ultrasound guidance is first line with a success of 75% to 95%. Irreducible intussusceptions will need surgical management, as will most intussusceptions in an adult.

### KEY POINTS

- AMS can be the predominant or only symptom of intussusception in a child.
- Positive results from FOBT and POC ultrasonography can increase the likelihood of intussusception.
- Negative results on a three-view abdominal radiograph can decrease the likelihood of intussusception.
- Radiology-performed ultrasound is comparable to air contrast enema for diagnosis, but enema remains the gold standard for diagnosis and treatment.

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# **DON'T MISS AORTOENTERIC FISTULA: A RARE BUT LIFE- THREATENING CAUSE OF GASTROINTESTINAL BLEEDING!**

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**KRISTIN BERONA, MD**

Aortoenteric fistula (AEF) is a rare cause of gastrointestinal bleeding (GIB); however, without treatment, mortality reaches 100%. Unfortunately, the classic triad of GIB, abdominal pain, and pulsatile abdominal mass occurs only 11% of the time.

Primary AEF refers to a spontaneous fistulous connection between the aorta and any part of the bowel. The most common cause (83%) of primary AEF is an aortic aneurysm, but infection, tumor, foreign body ingestion, and radiation therapy have all been reported. Primary AEF is exceedingly rare, with an incidence at autopsy of <0.1%.

Secondary AEF refers to formation of a fistulous connection after aortic surgery, occurring 0.5% to 2.3% of the time. It is much more common after emergent surgery for ruptured aortic aneurysm, but can happen after any aortic surgery including elective abdominal aortic aneurysm (AAA) resection, aortic replacement or bypass for aortoiliac occlusive disease, and open or endovascular stent graft placement for aortic aneurysm. It is often associated with aortitis: from the initial surgery, intestinal flora translocation, or postoperative bacteremia from another source. Median time from initial surgery to fistula occurrence is 2 to 4 years, though there have been reports of AEF occurring from 1 week to 26 years.

Both primary and secondary AEF have a distinct male predominance.

Fifty to seventy percent of cases are associated with the third and fourth portions of the duodenum due to its retroperitoneal placement and proximity to the aorta.

GIB is the most common presentation of AEF, occurring in >90% of patients. Hematemesis is most common, though melena and hematochezia have also been documented. Furthermore, patients may present with a small bleed that is self-limited due to thrombus formation and vasospasm. This initial bleed has been dubbed a “herald bleed” and occurs in about 50% of cases of AEF. A herald bleed may precede further bleeding anywhere from hours to weeks later. Patients may also present with nonspecific symptoms such as abdominal pain, back pain, fever, sepsis, or hypotension.

AEF should be considered and ruled out in any patient with a history of AAA or aortic repair who presents with GIB. Consideration of AEF is crucial to making the diagnosis, because the usual initial diagnostic procedures of endoscopy and colonoscopy in GIB will lead to the diagnosis of only 30% and 10% of patients, respectively. Even more frustratingly, 23% of patients with primary AEF in one series had concurrent gastric ulcers, which could lead to missing the diagnosis. Concern for AEF should be communicated to the gastroenterologist so he/she can make every effort to evaluate all the way down to the third or fourth portions of the duodenum.

If AEF is being considered and the patient is hemodynamically stable, CT of abdomen/pelvis with aortogram is the first-line diagnostic test, with a reported sensitivity of 94% and specificity of 85%. Extravasation of contrast from the aorta into adjacent bowel is a pathognomonic finding on CT, though it is rarely seen. Other suggestive signs include gas collections around aortic grafts, bowel wall thickening near an aortic graft, loss of the fat plane between aorta and intestine, and intramural duodenal hematoma. Again, communication with the radiologist regarding concern for AEF may help clinch the diagnosis.

If AEF is a strong consideration and the patient cannot be stabilized for a CT scan, prompt consultation with a vascular surgeon for exploratory laparotomy and possible repair is paramount.

Emergency department management of AEF follows the same principles as for any critical patient: at least two large-bore IVs, cardiac monitoring, and complete blood count (CBC) and chemistry, including renal function tests, PT/INR, and type and cross. Blood products and IV fluids are important for stabilizing a hypotensive bleeding patient; however, in AEF, one may consider controlled hypotension similar to trauma or ruptured AAA. A clot may be temporarily stopping the bleeding, so allowing the patient to remain relatively hypotensive prior to operation may be beneficial.

In addition to emergency stabilization, broad-spectrum antibiotics against gram-positive and gram-negative bacteria should be initiated. These will be continued for at least 1 week even after negative operative cultures and 4 to 6 weeks (tailored to culture sensitivities) in the case of positive cultures.

Definitive management is surgical, so it is important to involve vascular surgeons early in the management of patients with AEF. Hospitals without a vascular surgeon will need to stabilize and transfer these patients. Historically, and depending on the degree of infection, surgical AEF repair has involved open repair of aorta, graft placement, and closure of fistula or graft removal with axillobifemoral bypass. Thirty-day mortality is reported at about 30% to 40% after surgery, though one newer series from 1991 to 2003 reports a 21% 30-day mortality. Unsurprisingly, early death was significantly associated with patients presenting in hypovolemic or septic shock. In recent years, endovascular AEF repair (a minimally invasive interventional radiological technique) has been employed as a temporizing measure with lower postoperative mortality. Nevertheless, endovascular repair is currently limited by the complications of recurrent infection and bleeding—perhaps a reflection of the fact that the fistula is not primarily repaired.

## KEY POINTS

- Emergency providers must consider aortoenteric fistula in any patient with a history of aortic surgery or AAA who presents with GIB.
- Contrast CT is key to the diagnosis in stable patients.
- Prompt vascular surgery consult is paramount, especially in unstable patients.

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# ACUTE MESENTERIC ISCHEMIA: A TRUE ABDOMINAL CATASTROPHE

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TALIB OMER, MD, PhD, RDMS

Acute mesenteric ischemia (AMI) occurs when blood flow to the small intestines is not sufficient for normal bowel function. AMI is a high-risk diagnosis that carries an extremely high mortality rate (beyond 70%) if not recognized in a timely manner before bowel infarction occurs.

AMI can be divided into three different syndromes based on pathophysiology. It is important to distinguish these, as each one has different risk factors and different management strategies:

- 1) **Acute mesenteric arterial occlusion** is the most common (65% to 70%) and the most devastating cause with the highest mortality. It is mostly commonly encountered in elderly patients. The majority of these cases result from embolization of clots from the heart. The main risk factors are atrial fibrillation and myocardial akinesis, for example, as seen in an acute myocardial infarction. The remaining cases of mesenteric arterial occlusion occur due to an in situ thrombotic event in patients with severe atherosclerosis, mostly at the site of the proximal SMA. As the mechanism resembles that of a myocardial infarction, the main risk factors similarly are age, hypertension, and severe vascular disease. About half of these patients endorse a history of postprandial abdominal angina.
- 2) **Mesenteric venous thrombosis** (5% to 15%) is the only category of AMI that tends to affect younger patients. It has a similar mechanism as in other cases of venous thromboembolism (e.g., deep vein thrombosis and pulmonary embolism) and is likewise seen in patients with hypercoagulable states. In fact, the majority of patients with AMI

have a history of deep vein thrombosis. While the mortality rate is lower than in the other categories of AMI, it is still high at about 20% to 50%.

- 3) **Nonocclusive mesenteric ischemia** (20%) is a multifactorial condition often seen in hospitalized patients without evidence of a vascular occlusion. It can be caused by shock, a decrease in cardiac output, or medications (e.g., nodal blockers, vasoconstrictors) and can occur with or without narrowing of the mesenteric vasculature. It is associated with a high mortality, often due to the underlying condition.

The presentation of AMI varies based on the category, but the majority of patients complain of sudden onset of severe colicky, poorly localized abdominal pain that is often out of proportion to the examination findings. However, we cannot rely on this classically taught presentation of AMI. One of the main pitfalls of AMI is that some patients may have a seemingly benign examination and may initially only complain of mild diffuse abdominal discomfort. Abdominal distention and peritoneal signs are usually late findings that suggest that the bowel has already become necrotic. Septic shock and multiorgan dysfunction can develop well before, but bowel necrosis often occurs 10 to 12 hours after symptom onset. At this stage, mortality is about 70%.

Although often elevated, many laboratory studies like the white blood cell count, creatine kinase, phosphate, and amylase levels are neither sensitive nor specific enough to be helpful. An elevated lactate level has a good sensitivity; however, it may not pick up the early stages of the disease process, nor is it specific for AMI. Recently, the D-dimer assay has been promoted in the evaluation of AMI. Due to its high sensitivity, some authors suggest it could be utilized as a screening tool to rule out AMI.

The first-line imaging choice is CT angiography (CTA), though it only has a 12% to 15% sensitivity to directly visualize the lesion. Other CT findings include focal lack of bowel wall enhancement, bowel wall thickening, fat stranding, or pneumatosis intestinalis. Oral contrast should be avoided as it can obscure the view of the blood vessels. While CT is now considered first line given its availability, the gold standard remains conventional angiography for diagnosis and possibly therapy in the case of arterial occlusion.

Management includes intravenous (IV) fluid resuscitation, IV antibiotics, nasogastric tube placement, avoidance of nodal blockers and vasoconstrictive medications, and early surgical consultation. If vasopressors are needed for resuscitation, it is recommended that  $\alpha$ -agonists be avoided. If there is bowel necrosis on CT or peritoneal signs, immediate laparotomy is indicated, no

matter the cause of ischemia.

If no bowel necrosis is evident, specific management differs between the syndromes. If the cause is arterial occlusion, revascularization should be attempted with intra-arterial vasodilator infusion (e.g., papaverine) by interventional radiology or in some cases angioplasty, surgical thrombectomy, and thrombolysis. If the cause is venous thrombosis, the patient should be anticoagulated immediately (e.g., IV heparin), unless contraindicated. For AMI without evidence of vascular occlusion, the underlying condition should be treated; intra-arterial vasodilator infusion can be considered.

## KEY POINTS

- Consider AMI in elderly patients with a history of atrial fibrillation or vascular disease (even with a benign presentation and abdominal exam) or in younger patients with abdominal pain and a history of deep vein thrombosis (DVT) or hypercoagulable states.
- CTA is first-line imaging for diagnosis.
- Specific treatment varies based on etiology of AMI but includes prompt surgical consultation.
- Don't delay in the diagnosis and management of AMI: time is not only tissue, but time is mortality!

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# NOT ALL EPIGASTRIC PAIN IS BENIGN

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ALESSANDRA CONFORTO, MD

Although the incidence of peptic ulcer disease (PUD) has sharply declined, it is still estimated that 1 in 10 people in the United States is affected by PUD. Infection with *Helicobacter pylori* and medications such as nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin, and steroids are the most common culprits although acute stress (hospitalization, mechanical ventilation, and burns) and smoking also play a role. About 25% of patients with PUD will develop complications.

## UPPER GASTROINTESTINAL BLEEDING

PUD is the most common cause of acute upper gastrointestinal bleeding (UGIB). Although the majority of upper GI bleeds (85%) are self-limited, exsanguinating hemorrhage does occur and mortality is high (5% to 14%). Presenting symptoms include hematemesis, melena, or, if the rate/volume of blood loss is significant enough, hematochezia. Syncope, light-headedness, and generalized weakness can all be presentations of UGIB.

Signs of shock and abnormal vital signs must be addressed immediately and hemoglobin closely monitored: a normal initial hemoglobin does not rule out a significant bleed. Ongoing, large-volume hemorrhage is treated with rapid transfusion of red blood cells (RBCs), platelets, and fresh frozen plasma (FFP), usually in a 1:1:1 ratio. Early intubation for airway protection in this setting is key. Laboratory evaluation should include a complete blood count, metabolic panel, prothrombin time/international normalized ratio (INR), lactate, and type and screen.

In the absence of exsanguinating hemorrhage, the recommended

threshold for transfusion is a hemoglobin  $\leq 7$  mg/dL and potentially higher thresholds should be used in patients with severe cardiorespiratory comorbidities. Platelet transfusion is indicated for a platelet count of  $<50,000/\text{mL}$  (or  $<100,000/\text{mL}$  if platelet dysfunction is suspected). Mild to moderate elevation of INR ( $<1.5$ ) does not require correction, and endoscopy should not be delayed in order to correct an INR  $<2.5$ .

Nasogastric tube (NGT) insertion has not been shown to improve outcomes, and routine use is no longer recommended in clinical guidelines.

High-dose proton pump inhibitors (PPIs) (e.g., omeprazole 80 mg intravenous bolus followed by 8 mg/hour infusion) are recommended, though they have not been proven to decrease mortality, rebleeding, or the need for surgery. However, they do reduce the stigmata of hemorrhage at endoscopy.

Endoscopy is both diagnostic and therapeutic, providing definitive treatment of the bleeding ulcer in the majority of cases. Timing of endoscopy is predicated on the severity of the blood loss: immediately after resuscitation in patients with exsanguinating hemorrhage, within 24 hours for most patients, and as an outpatient procedure for those at extremely low risk. Of several scoring systems, the most useful in the emergency department setting is the Blatchford Risk Score. It defines patients at extremely low risk as those scoring  $<1$ ; these patients do not need admission and can be safely investigated as outpatients.

If endoscopy fails to control the active bleeding, other options include interventional radiology for embolization of the mesenteric artery and surgical intervention.

## PERFORATION

Perforation occurs if the ulcer erodes through the full thickness of the stomach or duodenal wall. Perforations may be contained (when adhesions, or other organs such as liver or pancreas prevent widespread leakage of gastroduodenal contents into the peritoneum) or free (unobstructed leakage of gastroduodenal contents into the peritoneum).

Perforation is more commonly associated with NSAIDs than *H. pylori* infection, although up to 10% of perforated gastric lesions are cancerous. Mortality is 5% to 10%: advanced age, comorbidities, immunodeficiencies, and cancer are poor prognostic factors.

Sudden-onset, severe, unrelenting, diffuse abdominal pain is the hallmark of perforation. The typical clinical picture consists of an ill-appearing patient, lying perfectly still, tachycardic, and likely hypotensive,

who has a fever and a rigid surgical abdomen with signs of peritoneal irritation and decreased bowel sounds. A more muted clinical presentation may occur in the elderly, immunosuppressed, or in patients with a contained perforation. An upright chest x-ray may reveal free intra-abdominal air (sensitivity 80%). The lateral view increases sensitivity to 98%, while CT of abdomen is even more sensitive and is able to detect gas collections of 5 mL.

Emergency department treatment includes IV fluid resuscitation; IV PPIs; broad-spectrum antibiotics to cover gram-negative bacilli, enterococci, and anaerobes; as well as NGT placement. In general, surgery is required to close the perforation, although medical management with antibiotics alone may be an option for stable patients with a contained perforation, spontaneously sealing perforations, or poor operative candidates.

## GASTRIC OUTLET OBSTRUCTION

Gastric outlet obstruction from PUD occurs infrequently but can be caused by marked edema, muscularis spasm, or irreversible narrowing due to scarring. It should be suspected in patients who present with recurrent vomiting that occurs several hours after eating. Symptoms also include anorexia, epigastric fullness, weight loss, and profound dehydration. The diagnosis can be made endoscopically or by CT scan.

Treatment includes NPO (nothing by mouth) status, NGT, IV fluids, and IV PPIs. If the obstruction does not resolve, endoscopy with balloon dilatation can be used as a temporizing measure. Definitive treatment is surgical with pyloroplasty, gastroenterostomy, or resection +/- vagotomy.

### KEY POINTS

- PUD can be a benign entity, but serious complications include UGIB, perforation, and gastric outlet obstruction.
- For UGIB: don't be lulled into a false sense of security by a normal initial hemoglobin and don't overtransfuse a stable patient. High-dose PPI and endoscopy are first-line therapies, with IR and surgery in life-threatening bleeds refractory to endoscopy.
- For perforation: normal CXR does not rule it out.
- For gastric outlet obstruction: consider this diagnosis; not all recurrent vomiting is gastroparesis!

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# **DON'T UNDERESTIMATE AN ACUTE VARICEAL HEMORRHAGE!**

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**LEE PLANTMASON, MD, MPH**

Upper gastrointestinal bleeding (UGIB) results from a variety of conditions that can vary from annoying to life threatening. The determination of nonvariceal versus variceal bleeding is critical as the tests and treatments vary depending on the etiology. Acute variceal hemorrhage (AVH) is the most common etiology of UGIB in patients with cirrhosis. It accounts for roughly 50% of cases and is associated with a mortality rate as high as 20%. Cirrhosis causes fibrotic changes in the hepatic parenchyma that decrease hepatic vascular compliance and increase portal vascular resistance. This in turn leads to dilatation of collateral vessels located at the gastroesophageal junction, that is, varices.

## **PRESENTATION**

Patients with a history of alcohol abuse, known cirrhosis, or a history of varices presenting with a UGIB should be presumed to have variceal bleeding and treated as such. Initial history taking should focus on the amount and route of hemorrhage. For hematemesis, is it coffee-ground or bright red? For blood by rectum, is it melena or hematochezia? Ask about other risk factors for UGIB such as a history of gastric/esophageal varices, use of anticoagulants and nonsteroidal anti-inflammatory drugs (NSAIDs), and other comorbidities that may affect the patient's ability to cope with acute blood loss. Symptoms of chest pain, shortness of breath, pallor, decreased urine output, or confusion may portend worsening end organ perfusion and shock.

## STABILIZATION

Given their potential to decompensate quickly, patients require large-bore intravenous access and close monitoring. Large-volume hematemesis, altered mental status, and hemodynamic instability are indications for early airway management due to risks of aspiration and to facilitate endoscopy. Circulatory collapse in the case of a brisk hemorrhage should be addressed with crystalloids and blood products with a target hemoglobin  $>7$  g/dL (potentially higher transfusion targets for those at risk of end organ dysfunction). Plasma or prothrombin complex concentrates (PCC) and platelets should be given to correct coagulopathy with a goal INR  $<1.8$  and platelets  $>50,000$ . However, remember, in rapid blood loss, there will be significant delay in the fall of the measured hemoglobin concentration, so treat the patient's hemodynamic and perfusion status, not the numbers from the laboratory!

Laboratory studies may include complete blood count (blood loss, platelets), coagulation factors (hepatic synthetic function), liver function tests (indication of hepatic dysfunction), basic metabolic panel (elevated BUN from blood in the alimentary canal and renal function), creatinine (renal function), and type and cross (transfusion). Bedside tests such as nasogastric lavage (NGL) and stool guaiac testing have classically been utilized in assessing an upper GI bleed. The utility of NGL, however, is questionable in all but the most severe cases of UGIB, and it causes great discomfort to patients.

## TREATMENT

Pharmacologic interventions for variceal bleeding include the use of proton pump inhibitors (PPIs), somatostatin analogs (octreotide), and antibiotics (ceftriaxone). PPIs are commonly used empirically in the cirrhotic with UGIB because brisk bleeding from peptic ulcer disease is also quite common. Current guidelines recommend a bolus of 80 mg of pantoprazole with an infusion of 8 mg/hour, but a recent meta-analysis in 2014 showed that intermittent bolus dosing was noninferior to bolus plus infusion for bleeding ulcers. Somatostatin analogues (namely, octreotide in the United States) are recommended to treat variceal bleeds to reduce portal hypertension. Octreotide is given as a bolus dose of 25 to 50 mcg IV with an infusion of 25 to 50 mcg/hour. To date, studies show an increased rate of early hemostasis and 5-day hemostasis but no change in adverse events nor a decrease in mortality. Prophylactic antibiotics (commonly ceftriaxone 1 g IV) should be given to all acute variceal bleeds because they confer a

mortality benefit in addition to decreasing rebleeding and hospital length of stay.

## CONSULTATION AND DISPOSITION

Early consultation with the gastroenterology service is recommended for immediate endoscopy, given that only 50% of variceal bleeding will stop on its own. Most patients with AVH will require ICU admission: indications include need for emergent endoscopy, hemodynamic instability or altered mental status, evidence of active bleeding (hematemesis or large bloody lavage), or significant comorbidities (coronary artery disease, cancer, alcohol withdrawal, etc.). Patients who fail endoscopic therapy may benefit from emergent transjugular intrahepatic portosystemic shunt (TIPS) to reduce portal pressure and achieve hemostasis.

Finally, in the case of the unstable patient without access to immediate endoscopy, balloon tamponade can be employed as a temporizing measure after the patient has been orotracheally intubated. Each commercially available balloon device has its own particular requirements for placement. The Sengstaken-Blakemore and Minnesota tubes have both a gastric balloon and an esophageal balloon. The Linton-Nachlas tube has only a single gastric balloon, which is usually sufficient to provide local tamponade of bleeding from gastroesophageal variceal bleeding. Complications can be severe and include esophageal rupture, airway obstruction, or aspiration pneumonia; however balloon tamponade is potentially lifesaving when other options are unavailable.

### KEY POINTS

- All patients with cirrhosis presenting with UGIB should be presumed to have AVH.
- These patients are sick and often require early advanced airway management and resuscitation with multiple blood products.
- Endoscopic therapy (consult GI early) + medical therapy (PPI, octreotide, and ceftriaxone) are first-line treatment.
- Consider TIPS or balloon tamponade for uncontrolled bleeding.

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# DON'T BE FOOLED BY A SUBTLE PRESENTATION—SBP CAN BE DEADLY!

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Every year, ~7% to 30% of patients with ascites develop spontaneous bacterial peritonitis (SBP), defined as the presence of infection in the ascitic fluid without any identifiable intra-abdominal source. It is thus distinct from peritonitis secondary to another intra-abdominal process (e.g., appendicitis) and from peritonitis that develops in patients receiving peritoneal dialysis. SBP is one of the most common sources of infection in patients with ascites, second only to urinary tract infections (UTIs). Unrecognized and untreated, SBP has a mortality of up to 90%! With early diagnosis and treatment, this improves dramatically; however, each hour's delay in antibiotic administration significantly increases mortality.

SBP occurs when bacteria translocate from the intestinal lumen to the ascitic fluid. The most common organisms are *Escherichia coli* (70%), *Klebsiella* species (10%), *Proteus* species (10%), *Enterococcus faecalis* (4%), and *Pseudomonas* species (2%). Cirrhotic patients have an unregulated release of inflammatory mediators in response to this bacterial load. The resulting cascade of sepsis pathways can quickly lead to multisystem organ failure and death.

Up to 70% of patients who have had one episode of SBP will go on to develop subsequent episodes within a year. Because of this, patients with one episode of SBP should receive lifelong *secondary* prophylaxis (commonly with an oral fluoroquinolone or trimethoprim-sulfamethoxazole). Two other groups of cirrhotic patients benefit from *primary* prophylaxis against SBP:

patients with acute variceal hemorrhage (who should receive antibiotic coverage for 7 to 14 days after the acute event) and those with decreased protein concentration in the ascitic fluid ( $\leq 10$  g/dL). Commonly used antibiotics include norfloxacin, ciprofloxacin, and trimethoprim-sulfamethoxazole.

Presenting signs and symptoms of SBP include fever, mild confusion (or worsening hepatic encephalopathy), diffuse abdominal pain, nausea, vomiting, decreased urine output, gastrointestinal bleeding, sepsis, and shock. Vague symptoms, unexplained leukocytosis, worsening renal function, and acidosis should all raise the concern for SBP in a patient with ascites. Patients with ascites will rarely have a rigid abdomen, so mild tenderness or pain should be taken seriously and investigated.

The presumptive diagnosis of SBP in the emergency department is based on peritoneal fluid cell count. The most commonly accepted criterion is the presence of  $>250$  PMN/mm<sup>3</sup> of ascitic fluid, although some authors use different cutoff values. If there is significant blood contamination ( $>10,000$  RBC/mm<sup>3</sup>), a correction factor subtracting 1 PMN for every 250 RBCs can be used. The use of a urine dipstick to detect elevated PMNs in ascitic fluid (via the leukocyte esterase test) allows for early presumptive diagnosis and antibiotic administration with improved outcomes. Gram stains are rarely positive because the concentration of bacteria is usually low. If blood culture bottles are inoculated at the bedside with at least 10 mL of ascitic fluid, diagnostic yield may be as high as 80%, but these results will not be available for several days after the initial visit. In addition to the cell count, the pH of ascitic fluid ( $<7.35$ ), a blood–ascitic fluid pH gradient  $\geq 0.10$ , as well as a serum-ascites albumin ratio  $\geq 1.1$  g/dL can help confirm the diagnosis of SBP in equivocal cases.

In contrast to SBP, secondary peritonitis should be suspected if ascitic PMNs are significantly  $>250$ /mm<sup>3</sup> or if two of the following criteria are present in ascitic fluid: glucose  $<50$  mg/dL, protein  $>10$  g/L, or ascitic LDH  $>$  serum LDH. The suspicion of secondary peritonitis mandates broadening antibiotic coverage to include anaerobic organisms and the search for a surgically correctable source of peritonitis with immediate surgical consultation and advanced imaging.

## TREATMENT

Third-generation cephalosporins (cefotaxime, ceftriaxone, or ceftazidime) for 5 days are the antibiotics of choice. Oral fluoroquinolones can be prescribed for patients who are awake and not vomiting. A repeat paracentesis should be

done only if there is inadequate response to treatment or secondary peritonitis is suspected. Albumin is recommended to decrease the incidence of SBP-induced renal failure on day 1 (1.5 g/kg) and day 3 (1 g/kg) of treatment in patients with Cr >1 mg/dL or total bilirubin >4 mg/dL.

## WHAT DO I DO IF I'M CALLED ABOUT A POSITIVE PERITONEAL CULTURE ON A PATIENT SENT HOME?

*Bacterascites* is a term used to describe an ascitic fluid that is colonized with bacteria (culture positive) but with a PMN count <250/mm<sup>3</sup>. The diagnosis is delayed, usually 3 days after the original paracentesis when the culture results become available. The current recommendations are to perform a repeat paracentesis on day 3. *Bacterascites* is treated as SBP if, on the second paracentesis, the PMN count is >250/mm<sup>3</sup>, or if the second culture is also positive. If PMN count is <250/mm<sup>3</sup> and cultures remain negative, no further action is necessary.

### KEY POINTS

- SBP can present with vague complaints, not necessarily with abdominal pain.
- The leukocyte esterase test on urine dip allows for early diagnosis and antibiotic administration.
- Patients who have had one episode of SBP are at risk for another and require lifelong prophylaxis.
- Don't mistake secondary peritonitis for SBP. If the ascitic fluid starts to resemble pus (very high PMN count), a surgical emergency should be considered.

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# ASCENDING CHOLANGITIS AKA BILIARY SEPSIS AKA “THAT OTHER PUS UNDER PRESSURE”

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**PRATHAP SOORIYAKUMARAN, MD**

A classic “you’ll miss it if it’s not on your differential,” ascending cholangitis refers to a bacterial infection of the biliary system, requiring both obstruction and bacterial colonization of the biliary tract.

Normally, bile is sterile. Bile salts have bacteriostatic properties, and the sphincter of Oddi controls the direction of bile flow, acting as a barrier between the sterile bile duct and the bacteria-filled duodenum. Without obstruction of the biliary system, ascending cholangitis does not occur. Even bacterial colonization of the bile in the absence of obstruction does not usually progress to clinical cholangitis. It’s still not always known how bacteria enter an obstructed biliary system, but one clear way is when the doctor does the dirty work by inadvertently interfering with the physiologic barrier between the bile duct and the intestine via surgery, endoscopic retrograde cholangiopancreatography (ERCP), or percutaneous transhepatic cholangiography (PTC). Bacteria, of which the most common are *Escherichia coli*, *Klebsiella*, *Enterococcus*, and *Bacteroides*, can also enter the biliary system from the lymphatics or via portal vein blood. Once the bacteria enter an obstructed bile duct, ascending cholangitis may result.

Who gets ascending cholangitis? You should think about it in any septic patient who has signs and symptoms of biliary tract disease (often subtle), especially if that patient is diabetic, elderly, or debilitated. Charcot was one of the first physicians to describe cholangitis, or “hepatic fever” as he called it, and he noted a constellation of symptoms that made up his triad:

intermittent fever with chills, right upper quadrant pain, and jaundice. Add mental status changes and shock, and you get Reynolds pentad, which confers a much graver prognosis without prompt decompression. The most frequent symptoms with acute cholangitis are fever and abdominal pain (approximately incidence of 80% in most reports). Clinical jaundice is less frequently seen (~60% to 70%). Severe presentations (e.g., with shock and altered mental status) are fortunately much less common (3.5% to 7.7%). Cholangitis rarely presents classically, so it is an important consideration in any septic patient without an obvious source. Importantly, the most severe cases are often the most difficult to detect as the patient may be too sick to help the clinician localize the infection of history and physical examination. Bedside ultrasound examination may be particularly helpful in this scenario as a screen for biliary pathology in the septic patient with altered mental status.

The causes of biliary obstruction are many. Common causes are outlined in [Table 87.1](#).

**TABLE 87.1 CAUSES OF BILIARY OBSTRUCTION**

- Gallstones
- Malignant biliary strictures (pancreatic cancer, cholangiocarcinoma, gallbladder cancer, ampullary tumor, duodenal malignancy)
- Benign biliary strictures (postsurgical, acute and chronic pancreatitis, primary sclerosing cholangitis, autoimmune cholangitis, congenital anomalies)
- Parasitic infections (higher index of suspicion in immigrants/travelers from endemic areas)
- Duodenal diverticulum (more common in older patients)
- Hemobilia
- Biliary stent obstruction

And don't forget, kids can get ascending cholangitis too! Kids who have undergone surgical Roux-en-Y procedures (such as the Kasai procedure for biliary atresia) and those with indwelling catheters or failure to thrive are at increased risk.

Once you suspect cholangitis, you'll be tasked with differentiating it from cholecystitis, both of which can present very similarly. An elevated bilirubin is more characteristic of cholangitis. Ultimately, though, ultrasound evidence of dilated common and intrahepatic bile ducts usually is required to distinguish cholangitis from cholecystitis.

The keys to treatment include hemodynamic stabilization, broad-spectrum antibiotics, admission to a monitored setting (ICU often), and

ultimately biliary tract decompression (surgery, interventional radiology, gastroenterology). These patients are sick, and if you don't make the diagnosis, they will not do well!

## KEY POINTS

- Consider in any septic patient without another source.
- Think of risk factors leading to obstruction of the biliary system, and include cholangitis in your differential for septic kids too.
- Differentiating cholangitis from cholecystitis: look for elevated bilirubin and ultrasound evidence of biliary duct dilation in cholangitis.
- ERCP can be the cause and the cure!

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# ACALCULOUS CHOLECYSTITIS: No STONES, No PROBLEMS?

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The diagnosis and epidemiology of acute calculous cholecystitis have been drilled into us since the early days of medical school. Every review book ever published has the mnemonic: Fat, Fertile, Forty, Family history, and Female. However, the discussion about acalculous cholecystitis has typically been short and inadequately summarized by: “it’s an ICU diagnosis in the critically ill.” The reporting and description of acalculous cholecystitis has evolved along with the advancement of its care. Duncan initially described it in 1844 in a patient who underwent a femoral hernia repair. Later, there was increased reporting and awareness during the Vietnam War in soldiers surviving sepsis and traumatic injuries. With the rise of intensive care units, the prevalence of acalculous cholecystitis also grew. Now that modern medicine allows patients to live longer with multiple comorbidities, we have even seen this disease in the outpatient setting. The key to diagnosis starts with an awareness of the unique set of risk factors these patients have (see [Table 88.1](#)).

**TABLE 88.1 RISK FACTORS ASSOCIATED WITH ACALCULOUS CHOLECYSTITIS**

| TRADITIONAL RISK FACTORS | RISK FACTORS FOUND IN ED PRESENTATIONS |
|--------------------------|--|
| ■ Burns                  | ■ Male                                 |
| ■ Trauma                 | ■ 60s                                  |
| ■ Nonbiliary surgeries   | ■ Hypertension                         |
| ■ Use of vasopressors    | ■ CAD                                  |
| ■ Mechanical ventilation | ■ Peripheral vascular disease          |
| ■ Childbirth             | ■ Diabetes                             |

Acalculous cholecystitis is a surgical emergency—a good prognosis hinges on early recognition and appropriate surgical treatment. It is estimated that acalculous cholecystitis comprises about 10% of all cholecystitis cases. In one study, within a 7-year period, 77% of all patients identified with acalculous cholecystitis presented in the outpatient setting. Importantly, 45% of those patients were ultimately admitted without the diagnosis of cholecystitis.<sup>1</sup> If specific treatment is delayed, this disease has a mortality rate as high as 65%, whereas, with early intervention, mortality is ~7%.<sup>2</sup> Contrast this with commonly reported numbers of 1.5% to 3% mortality for calculous cholecystitis.<sup>1</sup> Acalculous cholecystitis is a rare entity, but it is important to keep in mind in patients with sepsis due to an unclear source, especially in the setting of abdominal pain.

The pathophysiology of acalculous cholecystitis is thought to start with an acute or acute-on-chronic condition that leads to endothelial injury and gallbladder ischemia. The lack of perfusion creates stasis in the gallbladder, increased distention, and a localized inflammatory response. Some hypothesize that the bile salts and their by-products are toxic to the gallbladder when stasis occurs. Once cholecystitis has set in, a secondary bacterial infection can occur, usually with coliforms and anaerobes (commonly *Escherichia coli* and *Klebsiella pneumoniae*).<sup>2</sup> The exact nature and mechanism of the bacterial infection are not fully understood. Nevertheless, antibiotics are still considered a mainstay of treatment.

The clinical presentation can be varied, but most commonly, it consists of right upper quadrant abdominal pain, fever, jaundice, nausea, vomiting, and occasionally septic shock. Physical examination may find a palpable right upper quadrant mass and laboratory tests may show leukocytosis and elevated liver enzymes. Inpatients are more apt to have the major identifying risk factors: burns, trauma, nonbiliary surgeries, use of inotropes, mechanical ventilation, or childbirth. It is most certainly more difficult to pick these patients out in the outpatient and emergency department (ED) settings.

Acalculous cholecystitis is more common in males (1:1 to 2.8:1 male to female predominance), advanced age (average age is in the 60s), cardiovascular disease, diabetes, hypertension, peripheral vascular disease, alcoholic liver disease, and COPD.<sup>1,2</sup> These cases can be complicated by the patient being demented or developmentally delayed, which may prevent an adequate history and physical exam. In children, acalculous cholecystitis may present as a complication of Epstein-Barr virus (EBV) infection.<sup>3</sup> Patients with acquired immunodeficiency syndrome (AIDS) may also present with a nonspecific infectious prodrome and upper abdominal pain with acalculous cholecystitis secondary to an opportunistic infection, though it is more common for these patients to have cholangitis.

Diagnostic imaging is critical in making the diagnosis early. One study evaluating computed tomography (CT), ultrasound, and scintigraphy found that ultrasound (sensitivity 92%, specificity 96%) and CT (sensitivity 100%, specificity 100%) were both excellent diagnostic modalities but that scintigraphy suffered from poor specificity (38%).<sup>4</sup> Often, at the time of diagnosis, the differential remains broad and a CT scan may offer further diagnostic assessment for other possibilities on the differential.

On a positive note, once the diagnosis is made, the treatment remains similar to that of calculous cholecystitis. Antibiotics should be initiated to cover coliforms and anaerobes, typically a beta-lactam with a beta-lactamase inhibitor. Surgical consultation is necessary to decide whether the patient should be taken to the operating room for cholecystectomy versus an interventional radiology-guided cholecystostomy.<sup>1</sup> This decision usually revolves around the patient's comorbidities and perioperative risk. Complications such as emphysematous cholecystitis and perforated gallbladder can drastically alter the patient's course and treatment plan, further reinforcing the importance of early diagnosis in the ED.

## KEY POINTS

- Not all cholecystitis is caused by gallstones.
- Many patients with acalculous cholecystitis may present from the *outpatient* setting.
- Once the diagnosis is made, initiate broad-spectrum antibiotics and obtain early surgical consultation.

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# **ANTICIPATE BLEEDING AND REVERSE COAGULOPATHIES IN PATIENTS WITH LIVER FAILURE**

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Coagulopathy of liver disease leads to troubles as dramatic as large-volume hematemesis in a resuscitation bay or as indolent and irritating as a slowly oozing bleed around a central line on a boarded patient waiting for transfer out of the emergency department (ED). By evaluating, anticipating, and correcting bleeding diatheses when appropriate, the emergency physician (EP) can help the patient achieve hemostasis and avoid these untoward outcomes.

Blood clotting in the setting of chronic liver disease is complex and may result in a net prothrombotic or antithrombotic state. Favoring bleeding are reductions in hepatically produced procoagulant factors (II, VII, IX, and X) and fibrinogen, as well as thrombocytopenia due to reduced thrombopoietin production, splenic sequestration, and production of antiplatelet antibodies. Favoring thromboses are reductions in a strong anticoagulant factor (protein C) and increased platelet activation due to elevated von Willebrand factor levels. Complicating this situation is the lack of accurate lab assays to measure the net thrombotic state in liver disease patients. The international normalized ratio (INR) was validated as a standardized measure of anticoagulation for patients treated with Coumadin, but it is not calibrated to assess coagulopathy in liver disease patients. Indeed, the risk of venous thromboembolism in hospitalized chronic liver disease patients is higher than for hospitalized patients without liver disease, indicating that a prothrombotic



state is also frequently present. Despite these issues, transfusion of blood products to correct these coagulation abnormalities is recommended when active bleeding is present.

## EVALUATION OF COAGULOPATHY

Fortunately for the goal-oriented provider, a small number of crucial laboratory tests are needed for rapid coagulopathy assessment in the ED. Any patient with liver failure either actively bleeding or at risk of bleeding should have the following tests sent: prothrombin time/INR, fibrinogen level, and platelet count. A full battery of testing may help guide further evaluation and management down the road, but acute interventions will focus on these three tests.

## ANTICIPATING NEED FOR CORRECTION

Patients with liver failure generally compensate for coagulopathy, and corrective medications or blood products should not be administered to patients without an active or anticipated source of bleeding, such as planned invasive procedures. Acute correction is indicated for patients bleeding from gastrointestinal, traumatic, or iatrogenic sources. Procedures with a high risk of harmful bleeding include central lines, lumbar punctures, and intracranial monitor placement; the urgency of these procedures must be weighed against the risk of waiting for correction of coagulopathy. Lower-risk procedures such as paracentesis and peripheral vascular access do not necessitate correction.

## APPROPRIATE SELECTION OF TREATMENT

Treatment based on laboratory abnormalities is summarized in [Table 89.1](#).

|  |
|--|
| <b>TABLE 89.1 LABORATORY ABNORMALITIES AND TREATMENT</b> |
|--|

| LAB TEST         | TREATMENT THRESHOLD  | THERAPY  | COMMENTS            |
|------------------|--|--|---------------------|
| Platelet count   | 50 × 10 <sup>9</sup> /L with bleeding or high-risk procedure<br>100 × 10 <sup>9</sup> /L with very high-risk procedure<br>10 × 10 <sup>9</sup> /L with low-risk procedure or without active bleeding | 1 unit platelet transfusion (then reassess level before procedure if time allows, and administer additional platelets if below target level) |                     |
| Fibrinogen level | 100 mg/dL with bleeding or high-risk procedure   | Cryoprecipitate, weight based  | Typically 10 units  |
| INR              | >2.0 with bleeding or high-risk procedure  | FFP, weight based  | Typically 2–4 units |

Interventions based on the fibrinogen level and platelet count are fairly straightforward. If the fibrinogen level is under 100 mg/dL and the patient warrants correction, cryoprecipitate should be given with weight-based dosing. This is typically around ten units. If the platelet count is <50 × 10<sup>9</sup>/L during active or anticipated bleeding, 1 unit of platelets should be administered. Certain very high-risk procedures, such as ICP monitor placement, may warrant a higher platelet goal of >100 × 10<sup>9</sup>/L. For patients without active or anticipated bleeding, platelet transfusion should be considered only for a platelet count of <10 × 10<sup>9</sup>/L.

Treating coagulopathy based on INR is more complicated, due to the multifactorial nature of delayed hemostasis and lack of consensus recommendations. Fresh frozen plasma (FFP) is the frontline therapy for active or anticipated bleeding with INR over 2.0. Of note, the large volume of FFP required to treat coagulopathy may cause volume overload, which can worsen portal hypertension and bleeding due to gastroesophageal varices. Recombinant factor 7 may be considered in the case of life-threatening bleeding, but outcome-based research remains limited and this agent is extremely expensive. The role for other agents, including prothrombin complex concentrates, is currently under investigation.

Although total vitamin K deficiency is not directly caused by chronic liver disease, it frequently accompanies the malnourishment that occurs with chronic alcohol abuse. Administration of parenteral (intravenous, intramuscular, or subcutaneous) vitamin K should therefore be considered in alcoholic patients with active bleeding when the INR is elevated, regardless of whether liver disease is present.

Coagulopathy in patients with liver failure is complex due to imbalances

in both procoagulant and anticoagulant pathways. In the setting of active or anticipated bleeding, the EP should evaluate the PT/INR, fibrinogen, and platelet counts in these patients and prudently correct abnormalities based on the clinical scenario and the severity of bleeding or anticipated procedural risk.

## KEY POINTS

- If your patient has liver disease, anticipate both bleeding and clotting.
- Check PT/INR, fibrinogen levels, and platelet count in all patients at risk of bleeding.
- Correct coagulopathies judiciously and with appropriate therapy.

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# **BOERHAAVE SYNDROME: NOT ALL LIFE-THREATENING CHEST PAIN INVOLVES THE HEART AND LUNGS**

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Boerhaave syndrome is a spontaneous rupture of the esophagus. It usually results from barotrauma related to retching or any sudden increase in intra-abdominal pressure against a closed glottis. Esophageal perforations are rare and Boerhaave syndrome even rarer, accounting for only about 15% of cases. However, the mortality from this disease varies from ~8% to 60% and increases significantly if care is delayed. Although we have known about this syndrome for over 200 years, its associated morbidity and mortality remain high due to its nonspecific presenting symptoms and resulting delays in diagnosis.

Classically, we associate Boerhaave syndrome with retrosternal chest pain, vomiting, and subcutaneous emphysema (Mackler triad). Like with many other difficult diagnoses, these classic symptoms seldom present all together. Approximately 25% to 45% of patients have no history of vomiting, and even fewer have subcutaneous emphysema. The diagnosis of Boerhaave syndrome should be suspected in any patient who presents with retrosternal chest pain, neck pain, or epigastric pain, especially if it began after an episode of severe retching or bearing down against a closed glottis (weight lifting, defecation, childbirth, etc.). The location of pain generally correlates with the location of esophageal rupture, which most commonly occurs in the distal left posterolateral portion. Be aware that the severity of symptoms may vary widely depending on the time from the actual rupture, ranging from simple retrosternal pain, to respiratory collapse, septic shock, and multisystem organ failure.

The key to diagnosing Boerhaave syndrome is merely to consider the diagnosis. Many cases, however, are diagnosed incidentally on chest radiographs that reveal mediastinal air, subcutaneous emphysema, a pneumothorax, or a pleural effusion. Although the gold standard of diagnosis remains the contrast esophagram (with Gastrografin), computed tomography (CT) has become more readily available, and a chest CT can often suggest an esophageal perforation through the detection of esophageal edema, periesophageal fluid, or air in the mediastinal or pleural spaces. Additionally, if clinical suspicion is high, CT esophagography can be performed using diluted oral contrast to allow visualization of the specific site of perforation. Although endoscopy may aid in the diagnosis of Boerhaave syndrome, it is generally not recommended due to the risk of extending the esophageal tear through insufflation or direct trauma.

Once the diagnosis of Boerhaave syndrome is established, treatment should proceed rapidly. Even if a patient initially looks stable, decompensation may be rapid. Patients with Boerhaave syndrome should be regarded as critically ill and be monitored closely, especially in regard to their respiratory status. Many will present dehydrated and will require intravenous fluids while remaining NPO (nothing by mouth). Broad-spectrum intravenous antibiotics should be initiated early including coverage of enteric organisms. Intravenous proton pump inhibitors may also be considered, so as to decrease the severity of chemical mediastinitis and pleuritis.

Ultimately, the definitive treatment of Boerhaave syndrome is *rapid* surgical or endoscopic repair. A surgical consultation should be placed early once the diagnosis is suspected, given that early repair has been shown to decrease morbidity and mortality. Nevertheless, in certain subgroups of esophageal perforation, such as cervical perforation, or contained perforation with limited extraluminal extravasation of fluid, nonoperative management may be considered.

## KEY POINTS

- Boerhaave syndrome is a rare disease that is almost universally fatal if treatment is not performed.
- Consider this syndrome in *any* patient that presents with retrosternal pain.
- Recognize that these patients are sick (or soon will be).
- Initiate intravenous fluids and broad-spectrum antibiotics early in the

course of treatment.

- Consult surgery as soon as the diagnosis is suspected for expedited surgical or endoscopic repair.

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## CAUSTIC INGESTIONS: DON'T MAKE IT WORSE

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The 5,000 to 15,000 caustic ingestions (CI) per year in the United States occur in both children and adults. Eighty percent of these ingestions occur accidentally in children from 1 to 5 years old, and the rest typically occur intentionally in adults greater than 21 years old. Serious ingestions can immediately result in perforation, shock, and even death. Intentional ingestions in adults tend to have more serious consequences. Long-term complications can lead to strictures and an increased risk of esophageal cancer. In the emergency department (ED), we need to be aware of the atypical presentations of CI in children and be prepared for the immediate resuscitation of high-volume ingestions in adults.

Caustic materials cause tissue injury by chemical reaction. These materials are generally acidic or alkaline. The severity of tissue injury is determined by pH, concentration, duration of contact, amount, and physical form of the ingested substance. Acids cause coagulative necrosis, which results in a self-limiting burn pattern, while alkaline materials induce liquefactive necrosis with diffusion into deeper layers of the injured mucosa (see [Table 91.1](#)). Even low concentrations of alkaline ingestion can cause extensive injury.

**TABLE 91.1 ALKALINE AND ACIDIC CAUSTIC INGESTIONS**

|                  | <b>ALKALINE</b>  | <b>ACIDIC</b>   |
|------------------|--|---|
| pH of concern    | >11  | <3  |
| Type of necrosis | Liquefactive   | Coagulation   |
| Damage           | Hydroxy (OH) group reacts with tissue → swelling → small vessel thrombosis and heat  | Eschar forms, preventing deep tissue penetration → greater incidence of perforation   |
| Timeline         | Minutes  | Delayed   |
| Location         | Oropharynx<br>Hypopharynx<br>Esophagus   | Stomach   |
| Complications    | Acute and delayed perforation<br>Perforation<br>Strictures<br>Death  | Acute and delayed perforation<br>Hemorrhage<br>Small bowel exposure<br>Gastric outlet syndrome, metabolic acidosis<br>Hemolysis<br>Acute renal failure<br>Death                           |
| Examples         | Sodium hydroxide, lye (oven cleaners, liquid agents, liquid drain cleaners, disk batteries)<br>Calcium and lithium hydroxide (hair relaxers)<br>Ammonia (household cleaners) | Sulfuric acid, hydrochloric acid, nitric acid (toilet bowl cleaners, swimming pool cleaners, rust removers)<br>Hypochlorous acid (bleach—generally neutral pH), peroxide (mildew remover) |

CI can provoke injury from the mouth, the airway, down through the esophagus to the small intestine. Depending on the quantity, intent, and timing of the caustic ingestion, patients may present with a myriad of symptoms. Obvious burns to the lips, mouth, and oropharynx may occur, but do not be fooled if these signs are not present. Adults with intentional ingestions without any oropharynx involvement may have significant esophageal involvement (think about a fast intentional ingestion, where the liquid does not burn the oropharynx). Furthermore, laryngeal or epiglottic edema may present with stridor, dysphonia, hoarseness, dyspnea, and drooling, leading to respiratory distress and impending airway obstruction. Severe CI can cause esophageal perforation and may present with abdominal pain, rigidity, substernal chest pain, or back pain.

While the diagnosis is obvious when the history is clear, there are case reports of children presenting with symptoms of allergic reaction, being treated as anaphylaxis and later found to have a CI. Thus, in children presenting with allergic symptoms not improving with treatment, CI should be considered in the differential, as the initial presentation is similar and



otherwise can be easily missed. Unfortunately, even if the clinician considers CI, there is currently no definitive way to establish the diagnosis in the emergent setting if the ingestion is not reported. Radiographs can help provide information regarding perforation but are not always diagnostic. Only through direct visualization (usually by endoscopy) can the definitive diagnosis of CI be made. When performed within the first 72 hours after ingestion, endoscopy stages pathology and identifies the need for further intervention. If patients have any oropharyngeal injury, drooling, vomiting, dysphasia, or pain, a high-grade injury is likely, and urgent endoscopy should be performed to determine if surgical intervention is required.

The only management of CI that has reached consensus is to avoid agents that induce vomiting, such as ipecac, as this can lead to esophageal perforation. In the ED, the provider should attempt to identify the ingested product and the concentration of active ingredients. Further injury can result from attempting to neutralize the substance by using a weak acid or base and should be avoided. Moreover, diluents should not be used with any caustic ingestion. Because of poor adsorption and endoscopic interference, activated charcoal is also not given in caustic ingestions.

When managing CI, remember to evaluate the patient's airway first. Equipment for endotracheal intubation and cricothyrotomy should be readily available. If significant edema is present, consider fiberoptic-assisted intubation. Always place the patient NPO (nothing by mouth) until the extent of injury can be determined. If a suicide attempt is suspected, consider ethanol, salicylate, and acetaminophen levels as well as a psychiatric evaluation. In some cases, salicylate ingestion has been shown to independently cause stricture formation. With large-volume liquid acid ingestions, nasogastric tube suction may be beneficial, but its use needs to be weighed against the risk of esophageal perforation. Aggressive hydration and medications to decrease acid production are given to prevent reflux-associated injury, while steroids remain controversial.

## KEY POINTS

- Do NOT induce emesis, use ipecac, give charcoal, or attempt to neutralize the ingested substance by using a weak acid or base.
- In children: consider CI in patients who present with symptoms of anaphylaxis that do not improve with treatment.
- Endoscopy is indicated within 24 to 48 hours for any patient who is symptomatic (or asymptomatic with an alkali), children refusing to eat

or drink, or patients with altered mental status.

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## INGESTED FOREIGN BODIES: WHEN TO INTERVENE?

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Swallowed foreign body impaction occurs most commonly in children and edentulous or otherwise impaired adults. In adults, by far and away the most common foreign body ingestion is a meat bolus impacting a preexisting anatomic structure, and in children, the most common foreign body ingestion is coins. While most of these ingestions will pass spontaneously, nearly 20% will require endoscopy and 1% will require surgical removal. The manifestations are surprisingly broad and include the typical presentation of dysphagia and neck tenderness after eating, but can also include choking, wheezing, respiratory distress, or even a relatively asymptomatic patient. To make matters worse, this is a patient population that quite often is not able to provide an accurate history.

The esophagus naturally narrows at three places: the upper esophageal sphincter, the aortic arch, and at the diaphragm. All of these sites are common areas for impaction; however, many impactions, particularly recurrent ones, are secondary to webs, strictures, or masses. While an acute presentation is more common, partially obstructing lesions can present in a delayed fashion (days later) and are at higher risk for esophageal perforation.

The clinical history is not always sufficient. These patients are often elderly, are very young, or have a psychiatric diagnosis that would make their clinical history less reliable. In the vast majority of cases, a screening radiograph is appropriate. Be aware that many foreign bodies are not radiopaque! These include fish bones, most pills, and meat boluses. Therefore, a negative radiograph does not exclude a foreign body. In fact, approximately two-thirds of ingestions are radiopaque. There are a few tricks to reading these plain films that may be helpful:

- Disk batteries often appear to be a double shadow or coin stack.
- Tracheal foreign bodies are typically best seen on lateral projection.
- Esophageal foreign bodies are better seen in a coronal view.

The timing of intervention will vary depending on the clinical scenario, even with similar ingested objects ([Table 92.1](#)). The emergency medicine mantra of “the ABCs” still reigns here, and as with any other case, airway compromise is a true emergency. Other “hard signs” for emergent endoscopy include inability to handle secretions, fever, crepitus, or free air on radiograph. Furthermore, if the object is sharp, a disk battery, or a magnet, endoscopy should not wait. Cases to consider urgent endoscopy (rather than emergent) include large objects, inability to tolerate oral solids or liquids, and foreign bodies in the esophagus longer than 24 hours.

**TABLE 92.1 MANAGEMENT OF SPECIFIC INGESTIONS**

|                     |  |
|---------------------|--|
| Food bolus          | Glucagon 1 g IV. Endoscopy within 24 h<br><i>Note: Do not use meat tenderizer.</i>   |
| Sharp objects       | Consider CT scan to rule out perforation or abscess. If negative, consider endoscopy with full operating room backup; otherwise, consider surgical approach. |
| Disk batteries      | Emergent endoscopy or retrieval if in the esophagus; otherwise, watchful waiting.  |
| Magnets             | While only dangerous when more than one, given history limitations, it is best to remove them all.   |
| Round objects/coins | Forceps if visible, endoscopy if impacted, watchful waiting if through to the stomach and relatively small   |
| Drug packets        | Watchful waiting is often appropriate. Manipulation risks rupture.   |

Special consideration should be given to pediatric patients. Their ingestions are often more insidious, and the history from caregivers is paramount. A plain film is usually very helpful in these cases; however, avoid oral contrast as it does not add much information and runs a high risk of aspiration. Once the ingested foreign object has been localized and identified, as long as it is not acutely dangerous, the child can be discharged and followed with serial imaging from his or her pediatrician until passage. Follow-up is recommended for anyone who has presented with this complaint, as a thorough evaluation for the cause of the impaction will help prevent recurrence and possibly other complications. In some cases, the episode will lead to the unmasking of another more sinister diagnosis, such as a malignancy, that requires prompt intervention. Ultimately, the goal is to prevent perforation, which runs a considerable mortality risk, both acutely

and in the future.

## KEY POINTS

- Assess the airway first.
- Localization and identification of the foreign object will guide treatment.
- Disk batteries, magnets, and sharp objects usually require immediate intervention.
- Get follow-up for these patients.

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# SEVERE ACUTE PANCREATITIS CAN BE SNEAKY

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Acute pancreatitis is the leading cause of gastrointestinal (GI) related hospitalizations in the United States, accounting for over 280,000 hospitalizations each year. One-fifth of these cases are considered severe, carrying a mortality rate of around 10% or 25%, depending on whether these cases are sterile or infected, respectively.

Recognition of severe acute pancreatitis (SAP) is tricky, as many of the criteria, such as Ranson, Imrie-Glasgow, and APACHE II, are determined 24 to 48 hours after presentation. This renders them unhelpful in the emergency department (ED) time frame. Meanwhile, the CT severity index/Balthazar score is not available unless CT has been performed—and CT imaging early on in patients with a straightforward diagnosis of pancreatitis is actually discouraged (see [Chapter 94](#)). Finally, it should be noted that the degree of lipase elevation itself is not helpful in predicting severity of acute pancreatitis.

Early identification and aggressive management of SAP are linked directly to improved survival, leading to the idea of the “golden hours” of SAP, treating SAP similar to severe sepsis or a severe burn.

Two new scores have been proposed in recent years that are helpful in the ED diagnosis of SAP. The first is the Bedside Index of Severity of Acute Pancreatitis (BISAP) score, which consists of five factors: blood urea nitrogen (BUN) >25 mg/dL, impaired mental status (Glasgow Coma Score <15), presence of systemic inflammatory response syndrome (SIRS), age >60 years old, and pleural effusion on imaging. Each variable, if present, is worth 1 point. Scores of 3, 4, and 5 are reflective of in-hospital mortality

rates of 8.3%, 19.3%, and 26.7%, respectively. A BISAP score  $\geq 3$  is also associated with an increased risk of developing organ failure, persistent organ failure, and pancreatic necrosis.

A scoring system that *excludes* SAP is the harmless acute pancreatitis (HAP) score. This score looks at three factors: hematocrit (Hct), serum creatinine (Cr)  $>2.0$  mg/dL, and rebound tenderness/guarding on exam. Patients without any of the three are unlikely to develop SAP (sensitivity of 96%). Independently, Cr  $> 1.8$  mg/dL is a predictor of poor prognosis in SAP.

Using these scoring systems alongside clinical experience, one has a better chance at identifying SAP. Severe acute pancreatitis can be a rapidly progressive disease and thus requires aggressive initial management in the ED even if a patient appears clinically well.

Fluid resuscitation and supportive care are the crux of early SAP management.

Common pitfalls include insufficient fluid resuscitation and using the wrong type of fluid. An initial bolus of 20 mL/kg followed by 3 mL/kg/h is appropriate with a goal urine output of 0.5 to 1 mL/kg/h, heart rate  $<100$ , systolic blood pressure  $>90$ , if there are no cardiac or pulmonary contraindications. A reassessment of BUN, Cr, lactate, and Hct should be performed in 6 to 8 hours, and at that time, the fluids should be decreased to 2 mL/kg/h if all the parameters are improving. Some evidence favors lactated Ringer's over normal saline, but this is an area of ongoing controversy. Prophylactic antibiotics are no longer recommended. However, if the patient has evidence of infection and/or meets SIRS criteria, empiric antibiotics are indicated.

For SAP, ICU admission should be discussed. At a minimum, surgery and GI should be consulted. RUQ ultrasound should be considered if gallstone pancreatitis is suspected. A contrast CT of the abdomen/pelvis can help further stage pancreatitis, but a CT is often more useful 48 to 72 hours after the onset of symptoms, after a trial of medical management, to detect surgical complications.

As fluid resuscitation continues, serial abdominal exams with measurement of abdominal compartment pressure should be conducted to ensure that abdominal compartment syndrome (ACS) does not develop. This is defined as an intra-abdominal compartment pressure of  $>20$  mm Hg associated with new-onset organ failure. If ACS develops, initial (medical) management includes

- 1) Decreasing intestinal volume: nasogastric/orogastric drainage,

promotility agents, rectal tubes, and, if necessary, endoscopic decompression

- 2) Decreasing intra-/extravascular fluid: decreasing volume resuscitation and, if volume overloaded, either ultrafiltration or diuretics
- 3) Medical abdominal wall expansion: analgesia and sedation to decrease abdominal muscle tone and, if necessary, neuromuscular blockade

If these strategies fail, surgical decompression may be indicated. However, new literature considers dialysis/hemofiltration as a less invasive alternative. Early surgery and nephrology consultations will assist with managing this insidious yet deadly complication.

In summary, SAP must be diagnosed early in the ED, treated aggressively, and monitored closely to minimize morbidity and mortality (see [Table 93.1](#)).

**TABLE 93.1 APPROACH TO SEVERE ACUTE PANCREATITIS**

|   |
|---|
| <b>Diagnosis:</b> BISAP score (BUN >25 mg/dL, GCS <15, SIRS, age >60, pleural effusion) + end organ damage + assessment of pH + HAPS criteria (rebound/guarding, increased Hct, elevated serum Cr >2.0 mg/dL) |
| <b>Labs/Imaging:</b> CBC, CMP, lactic acid, lipase, LDH, ABG/VBG (for pH), blood cultures × 2, triglycerides, CXR, consider RUQ ultrasound, consider CT abdomen/pelvis with contrast vs. no contrast          |
| <b>Treatment:</b> Fluid resuscitation goals: HR <100, SBP >90 mm Hg, BUN <22 mg/dL, UOP ≥0.5–1 mL/kg/h, Hct (improving hemoconcentration)   |
| <b>Fluid Dosing:</b> Bolus 20 mL/kg, then at least 3 mL/kg/h, reassess in 6–8 h   |
| <b>Fluid Type:</b> Lactated Ringer > normal saline  |
| <b>Antibiotics:</b> No prophylactic antibiotics BUT yes if evidence of infection/SIRS/sepsis  |
| <b>Complications:</b> Abdominal compartment syndrome, infected pancreatitis, pseudocyst formation, necrotizing pancreatitis, pancreatic duct strictures, loss of pancreatic function, death                   |
| <b>Consult:</b> ICU, surgery, GI, nephrology (if severe acidosis, worsening renal failure, abdominal compartment syndrome)  |
| <b>Consider:</b> Early dialysis/hemofiltration for acidosis and abdominal compartment syndrome  |

## KEY POINTS

- Early identification and aggressive management of SAP are linked directly to improved survival.
- Patients with SAP may develop abdominal compartment syndrome, infection, pseudocyst, and other life-threatening complications.



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# USE RESTRAINT WHEN IMAGING PATIENTS WITH ACUTE PANCREATITIS

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Acute pancreatitis is frequently encountered in the emergency department (ED) and results in hospital admission around 65% of the time in the United States. Many scoring systems have been developed to assess prognosis and severity of acute pancreatitis, often incorporating CT and MR imaging of the abdomen. However, this imaging is most beneficial later in the inpatient hospitalization rather than during the acute ED evaluation, unless the diagnosis is in question. Therefore, discretion should be used when ordering abdominal imaging for patients with a secure diagnosis of acute pancreatitis at the initial presentation.

## **PRESENTATION OF ACUTE PANCREATITIS**

Acute pancreatitis is common in the ED, but the presentation may be variable. Patients may suffer recurrent bouts and could be well known to ED staff with loud complaints of unmanaged pain and nausea while eating Flamin' Hot Cheetos® in the waiting room. Other patients may suffer more indolent symptoms that can be difficult to differentiate from dyspepsia. Severe acute pancreatitis can mimic sepsis due to severe inflammation, and patients may present with fever and shock due to extensive third spacing of intravascular volume. The most common etiologies of acute pancreatitis, gallstones, and alcohol consumption should be elicited as possible triggers.

## UTILITY OF IMAGING FOR DIAGNOSIS

Serum lipase levels have excellent sensitivity and specificity for pancreatitis in the acute phase of illness, approaching 95% in the first two days of symptoms. However, this level becomes more unreliable in patients with chronic or intermittent pancreatitis. In patients with typical symptoms and diagnostic laboratory tests, CT or MR imaging is not required to confirm the diagnosis of acute pancreatitis. Biliary imaging, typically by ultrasound, and social history for alcohol use are critical first steps to determine the underlying cause. When the diagnosis is unclear due to equivocal laboratory findings and atypical presentation, CT imaging of the abdomen and pelvis may be indicated to assess for nonpancreatitis causes of abdominal pain or to verify pancreatic inflammation confirming an acute pancreatitis diagnosis.

## UTILITY OF IMAGING FOR INTERVENTION AND PROGNOSIS

CT or MR imaging for acute pancreatitis is primarily used to detect complications such as pancreatic necrosis or fluid collections (pseudocysts) that may warrant interventional drainage. However, these findings are typically delayed by at least several days after the onset of symptoms. Moreover, the initial management of pancreatitis is supportive. When complications are detected on imaging, intervention is usually delayed until a week or longer after diagnosis and is reserved for fluid collections with mature cyst walls that do not resolve spontaneously or pancreatic necrosis with associated infection. While the optimal timing of imaging is unclear, it is generally recommended for patients with persistent symptoms or fever after 48 to 72 hours of medical management. Boarding times in the ED seldom approach these thresholds, so CT or MR imaging is not typically indicated in the ED for the patient with an established diagnosis of acute pancreatitis.

## HAZARDS OF IMAGING

A recent retrospective study at an academic hospital found that over half of all patients presenting to the ED with acute pancreatitis underwent CT imaging in the first 24 hours of admission; the vast majority of these patients had a clear diagnosis of acute pancreatitis without this imaging. Other studies have also demonstrated increasing rates of early or ED-based imaging of acute pancreatitis nationwide. Projections of radiation risk are dependent on many factors, but modeling predicts a number needed to harm of roughly

1,000 abdomen/pelvis CT exposures in healthy 40-year-old patients for each radiation-induced cancer. Detection of clinically insignificant lesions elsewhere in the abdomen that require additional invasive testing or repeat imaging (so-called incidentalomas) confers additional risk to the patient and draws excess resources and cost from the health care system. While early CT or MR imaging may be occasionally necessary in very ill patients or in cases where there is diagnostic uncertainty, routine imaging is seldom useful and potentially harmful. Be judicious with the radiation exposure risk of excess CT imaging.

## KEY POINTS

- CT/MR imaging is not indicated for assessment of clear-cut pancreatitis in the ED.
- Excess imaging carries real risk to the patient.
- Consider cross-sectional imaging if the diagnosis is unclear.

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# THE “PAIN” IN CHRONIC PANCREATITIS

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**DELPHINE J. HUANG, MD, MS**

Chronic pancreatitis occurs when there is irreversible and progressive destruction of the pancreas.<sup>1</sup> Patients frequently present with disabling chronic abdominal pain with those with intractable pain requiring large doses of pain medications.<sup>2,3</sup> Due to the loss of pancreatic function, patients may experience steatorrhea, malabsorption, weight loss, anorexia, nausea, vomiting and diabetes mellitus.<sup>3</sup>

The transition from acute to chronic pancreatitis can be indistinct and difficult to discern. Moreover, the clinical picture of chronic pancreatitis can be highly variable.<sup>2,3</sup> Clinicians frequently feel conflicted about doing a detailed workup as many of these patients are frequent users of the emergency department (ED) and may have opioid dependency issues. Nonetheless, it is important to assess a patient’s abdominal pain, as well as for complications of chronic pancreatitis.

In a patient with acute pancreatitis, lipase levels will most frequently be elevated. These enzymes levels can normalize as the disease progresses. In chronic pancreatitis, clinicians may be falsely reassured when they see a normal lipase, despite true pancreatic functional burnout.<sup>2</sup> Further, patients who do not consume any alcohol and present with chronic pancreatitis should be screened for gallstones and other toxic-metabolic, autoimmune, and genetic etiologies. To confirm the diagnosis in those with suspicious presentations, abdominal CT or MRI may be used to image the pancreas. In some cases, patients may need referral for an endoscopic ultrasound (EUS) or magnetic resonance cholangiopancreatography (MRCP), especially for mild or early disease states.<sup>2</sup> Once a diagnosis is established, however,

repeated imaging on subsequent presentations to the ED is not necessarily indicated.

While it may be easy to dismiss patients with chronic pancreatitis as “opiate seeking,” it is important to consider whether complications of this disease may be contributing to their acute exacerbation. For example, are there any new features, such as a change in the nature of the pain? Is a fever present? Patients with intractable vomiting, or who are unable to tolerate any food or fluids, may require further evaluation. Strictures of the pancreatic ducts, pseudocysts, pancreatic stones, and fistulization are common in this population. Fluid collections and pancreatic necrosis can develop into infections or abscesses that require antibiotics or drainage.<sup>2</sup> Diabetes mellitus, secondary to a loss of islet cells, can manifest in the later stages of disease. These patients may develop neuropathy and retinopathy and rarely, may even develop diabetes ketoacidosis. Lastly, the risk of pancreatic cancer is much higher in this population, especially in patients with hereditary pancreatitis.<sup>1–3</sup>

The ED management of pancreatic pain can be very challenging as therapeutic options may be limited. While the etiology of the pain is not well understood, it is most likely due to chronic inflammation, altered nociception, and tissue ischemia.<sup>1</sup> Acetaminophen is considered to be a first-line therapy, but by the time these patients arrive to the ED, they may require or request opioid medications. Other nonnarcotic options are frequently used in the outpatient setting. These include neuropathic pain modulators such as antidepressants (e.g., cyclic antidepressants and serotonin reuptake inhibitors), as well as, gabapentin. All have been shown to be effective.<sup>4</sup> More recently, some success has been demonstrated with the use of oral and intravenous ketamine, and this agent continues to be studied.<sup>5</sup>

Harm reduction counseling is extremely important. While not necessarily reversible, alcohol abstinence has been shown to slow or halt disease progression, which may improve chronic pain.<sup>3</sup> Cigarette smoking is also a major risk factor and contributor to the morbidity of chronic pancreatitis. Patients can also diminish their pain by eating smaller, low-fat meals; adding a proton pump inhibitor; and supplementing their diet with pancreatic enzymes and micronutrients to improve digestion.<sup>1</sup>

In patients with intractable pain, referral for a nerve block, endoscopy, or surgery should be considered.<sup>6</sup> Ductal decompression, lithotripsy, sphincterectomy, or pancreatectomy may all play a role in these cases to improve pain control. Islet cell transplantation also has been shown to be successful.<sup>2,3</sup> Lastly, corticosteroid therapy for those with autoimmune

disease can be beneficial.<sup>1</sup>

## KEY POINTS

- Lipase levels may be normal, despite true functional pancreatic burnout.
- Remember to assess patients for complications if pain cannot be controlled. These include pancreatic pseudocyst, necrosis, stricture, and abscess.
- If complications are not present and pain is not well controlled, consider referring patients for specialist consultation with a gastroenterologist, surgeon, or pain management specialist.

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# **ABDOMINAL PAIN IN INFLAMMATORY BOWEL DISEASE: A FLARE OR EMERGENT COMPLICATION?**

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Inflammatory bowel disease (IBD) includes Crohn disease (CD) and ulcerative colitis (UC). Crohn's disease is characterized by transmural bowel wall inflammation that can occur anywhere throughout the gastrointestinal tract. Normal mucosa surrounds diseased segments, resulting in characteristic "skip lesions." In contrast, ulcerative colitis is limited to the colon and typically only involves the superficial mucosal layer, but with continuous lesions starting from the rectum. In both CD and UC, fistulae, strictures, and abscesses can develop, leading to complications such as intestinal obstruction, perforation, infectious colitis, and toxic megacolon.

The exact pathophysiology of IBD is not well understood, but it is thought to be multifactorial, with a combination of genetic, environmental, and immune factors. In some cases, food-borne illnesses and increased intestinal wall permeability have been identified as inciting factors. In addition, bacterial, viral, or parasitic superinfections can trigger acute flares in IBD patients.

Patients with IBD typically present to the emergency department with abdominal pain and/or distention, along with bloody diarrhea. Fever, nausea, vomiting, fatigue, and weight loss are also common presenting symptoms. Symptoms may be acute and persistent ( $\geq 4$  weeks) or recurrent ( $\geq 2$  episodes in 6 months).



An abdominal series x-ray can be a useful screening tool to rapidly identify complications of IBD. The presence of free air, air-fluid levels, and/or dilated bowel loops with a paucity of distal bowel gas should alert the clinician to the possibility of bowel obstruction, perforation, and/or toxic megacolon. In adults and children  $\geq 10$  years, acute dilatation of the transverse colon to  $>5$  to 6 cm with the loss of haustral folds is diagnostic for toxic megacolon. In children  $<10$  years, a transverse colonic diameter of  $>4$  cm is suggestive. Toxic megacolon is a surgical emergency; it is associated with an increased risk of intestinal perforation and hemorrhage, electrolyte abnormalities, and sepsis.

In addition to plain x-ray, computed tomography (CT) imaging of the abdomen should be considered to look for other complications, such as abscess, stricture, and/or fistulae. CT imaging can also provide further details on intestinal obstruction and the extent of colitis. In pediatric patients, both ultrasound and magnetic resonance imaging are alternative imaging modalities that can be used to avoid ionizing radiation exposure, although such techniques are less well established and their use is limited by center-specific availability and expertise.

It is often challenging to distinguish whether a patient is having an acute flare or complications of IBD. Presenting symptoms are similar in both cases, as are results from laboratory testing. Such common laboratory abnormalities include thrombocytosis, anemia, and elevated inflammatory markers (C-reactive protein and erythrocyte sediment rate). Additionally, the use of corticosteroids or other immune modulators may potentially mask signs and symptoms of an acute abdominal emergency, leading to delayed diagnosis and treatment. A complication should be suspected in patients who have unstable vital signs, escalating abdominal pain, a toxic appearance, and/or signs of peritonitis.

Acute IBD flares require high-dose intravenous corticosteroids. Methylprednisolone 1 mg/kg q12h (with a maximum of 30 mg q12h) is the recommended first-line therapy. Patients should remain NPO for bowel rest, and intravenous fluids should be initiated.

Antibiotics are indicated when infectious colitis, perforation, and/or toxic megacolon are suspected. A combination of ciprofloxacin and metronidazole is typically the first-line therapy, but the choice of antibiotics should be tailored to the specific underlying infectious organism when results are available. While ciprofloxacin is not routinely used in pediatric patients younger than 12 years, it is an accepted alternative when no other effective and safe therapies are available and benefits outweigh risks. In more severe disease, surgical intervention may be indicated. Management decisions

should be made in consultation with gastroenterology and/or surgery.

As bacterial, viral, or parasitic superinfections can precipitate IBD flares, a stool sample should be cultured for enteric pathogens, including *Campylobacter*, *C. difficile*, cytomegalovirus, *E. coli*, *Entamoeba*, *Giardia*, *Salmonella*, *Shigella*, and *Yersinia* spp. Among these enteric pathogens, *C. difficile* is the most common and has been linked to worse outcomes.

The use of opioids is controversial in patients with IBD, as it has been associated with intestinal perforation and toxic megacolon, particularly with concurrent colitis. Other medications for pain management can be considered; studies have shown that ketamine (0.1 to 0.5 mg/kg) provides effective analgesia and decreases the opioid dose requirement. Benzodiazepines have also been shown to be effective for pain relief in patients with tenesmus.

## KEY POINTS

- Common complications of IBD include fistulae, strictures, abscesses, intestinal obstruction, perforation, infectious colitis, and toxic megacolon.
- Abdominal series x-ray can rapidly identify many complications of IBD and can be obtained prior to CT or other imaging modalities.
- The use of corticosteroids or other immune modulators in patients with IBD leads to immunosuppression and can obscure the diagnosis of an acute abdominal emergency. Serial abdominal exam is crucial.
- While antibiotics are only indicated in suspected infectious colitis, perforation, and toxic megacolon, their administration should not be delayed in toxic-appearing patients with escalating abdominal pain.

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# NOT EVERY PREGNANT PATIENT WITH VOMITING HAS HYPEREMESIS GRAVIDARUM

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Nausea and vomiting are common symptoms during pregnancy. It is estimated that between 50% and 90% of women will experience nausea and vomiting during their pregnancy, with 35% experiencing clinically significant vomiting. Nausea and vomiting usually begin in the first trimester and peak at 9 weeks of gestation, with more than 90% of cases resolving by the 20th week of gestation; this appears to be related to human chorionic gonadotropin and estradiol levels. Although common in otherwise normal pregnancies, we need to resist the temptation to immediately attribute nausea and vomiting to the physiology of pregnancy or to hyperemesis gravidarum. We should maintain a cautious and thoughtful differential diagnosis.

Hyperemesis gravidarum is considered the extreme end of a spectrum of nausea and vomiting in pregnancy. It is notable that hyperemesis gravidarum is present in only 0.5% to 2% of all pregnancies. This diagnosis requires exclusion of other causes and presents with a measure of actual starvation, manifesting as large ketonuria. Patients may also have electrolyte imbalance and dehydration and commonly have at least a 5% loss of prepregnancy weight. Regardless of etiology, when severe, vomiting can itself result in damage to both the mother and the fetus. Cases of splenic avulsion, esophageal rupture, pneumothorax, acute tubular necrosis, Wernicke encephalopathy, and central pontine myelinolysis have all been reported.

A careful history including a review of chronic medical conditions that

existed prior to the pregnancy, in addition to a targeted physical examination, will yield important clues to potentially serious causes. It should be noted that nausea and vomiting of pregnancy is rarely associated with other concerning symptoms such as fever, headache, neurologic deficits, abdominal pain, proteinuria, dysuria, hematuria, or flank pain. The presence of any of these symptoms should prompt an aggressive search for another etiology. [Table 97.1](#) gives a differential diagnosis for nausea and vomiting in pregnancy.

**TABLE 97.1 DIFFERENTIAL DIAGNOSIS OF NAUSEA AND VOMITING IN PREGNANCY**

**Gastroenterologic (GI) Conditions**

- Achalasia
- Biliary tract disease
- Hepatitis
- Gastroenteritis
- Gastroparesis
- Intestinal obstruction
- Peptic ulcer disease
- Pancreatitis
- Appendicitis

**Genitourinary (GU) Conditions**

- Pyelonephritis
- Uremia
- Ovarian torsion
- Renal stones
- Degenerating uterine leiomyoma

**Miscellaneous**

- Drug toxicity
- Drug withdrawal
- Drug intolerance

**Metabolic Disease**

- Diabetic ketoacidosis
- Porphyria
- Addison disease
- Hyperthyroidism

**Neurologic Disorders**

- Pseudotumor cerebri
- Vestibular lesions
- Migraines
- CNS tumor

**Pregnancy-Related Conditions**

- Acute fatty liver of pregnancy
- Preeclampsia
- Molar pregnancy

Adapted from Practice Bulletin No. 153: Nausea and Vomiting of Pregnancy. *Obstet Gynecol.* 2015;126(3): 687–688.

Evaluation should include blood work to check for electrolyte abnormalities as well as liver function tests, urinalysis, and a pelvic ultrasound to exclude molar pregnancy. After serious conditions have been excluded, the primary approach is to control symptoms through lifestyle and diet interventions. Recommend avoiding triggers of nausea (i.e., strong

smells, fatty or spicy foods, iron tablets, etc.). Small meals should be eaten frequently, and bland, dry, and high-protein diets appear to be better tolerated. Ginger and pyridoxine (vitamin B<sub>6</sub>) have been shown to be more effective than placebo. Both are considered safe in pregnancy and available over the counter.

When lifestyle and dietary changes have failed and over-the-counter therapies are ineffective, prescription medications can be considered. The American College of Obstetrics and Gynecology (ACOG) recommendations for initial management include pyridoxine hydrochloride 10 to 25 mg orally three to four times daily and doxylamine succinate, an antihistamine, at a dose of 12.5 mg orally three or four times daily. These are considered safe for mother and fetus. Failing this, second-line agents include diphenhydramine and/or promethazine. These are both antihistamines as well, so beware of compounding doses with previously prescribed doxylamine. Resistant cases can be treated with 5-HT<sub>3</sub> antagonists (ondansetron). Early studies have shown these agents to be generally safe in pregnancy, though one retrospective study has shown an increased risk of cardiovascular defects.

## KEY POINTS

- Although physiologic nausea and vomiting of pregnancy is common, always consider other underlying causes.
- Be familiar with lifestyle and dietary changes that may improve nausea without pharmacologic treatment. These are first line.
- Pyridoxine and doxylamine succinate are considered safe pharmacologic interventions.

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## BEWARE OF THE PATIENT WITH PAINLESS JAUNDICE

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Jaundice is defined as a yellowish discoloration of the skin and mucosa. It is the clinical manifestation of elevated serum bilirubin, which is largely the product of degraded red blood cells. Hyperbilirubinemia can be due to unconjugated bilirubin before it undergoes glucuronidation in the liver, or conjugated bilirubin after it has undergone glucuronidation and is excreted in stool (stercobilin) or urine (urobilinogen).

Important historical features in a patient with painless jaundice include an occupational history, history of toxin exposures, overseas travel, family history, a history of alcohol or intravenous (IV) drug abuse, or high-risk sexual activity. Significant weight loss, fevers, night sweats, and increasing abdominal girth can lead the physician to specific diagnoses such as parasitic infections, autoimmune diseases, or neoplasm. Signs and symptoms of anemia, a history of melena, and any other suggestion of gastrointestinal (GI) bleeding are very important and should be sought in the history.

On physical examination, there are certain findings that, in conjunction with jaundice, are concerning for an underlying *emergent* condition. These include any vital sign abnormalities and anemia, easily determined by bedside testing. Avoid the temptation to attribute abnormal vital signs to an underlying chronic disease. Any degree of hypotension, for example, should prompt an active search for a source of bleeding or infection.

Initial testing should include serum glucose, a complete blood count (CBC), electrolytes, liver function tests (LFTs), type and screen, and fecal occult blood. If the bilirubin level is abnormal, one must distinguish between

conjugated (direct) and unconjugated (indirect) bilirubin. GI bleeding is a critical consideration and should be recognized as quickly as possible; portal hypertension due to liver dysfunction increases the risk of gastroesophageal varices and ulcers that may manifest initially with hemorrhage (acute or chronic).

Jaundice with unconjugated hyperbilirubinemia in the presence of normal transaminases and alkaline phosphatase should raise concern for hemolysis. This may be drug-induced, autoimmune, or due to an inherited disorder (see [Table 98.1](#)). If hemolytic anemia is suspected, a peripheral smear should be ordered as well as reticulocyte count, haptoglobin, and lactate dehydrogenase (LDH). A hematologist should be consulted, and admission is warranted.

**TABLE 98.1 COMMON CAUSES OF HEMOLYTIC ANEMIA**

| <b>INFECTIONS</b>  | <b>EXTRINSIC</b>            | <b>ORGAN<br/>DEPENDENT</b> | <b>RBC INTRINSIC<br/>DEFECT</b> | <b>MICROANGIOPATHIC</b>               |
|--------------------|-----------------------------|----------------------------|---------------------------------|---------------------------------------|
| Malaria            | Transfusion reactions (ABO) | Liver failure              | G6PD deficiencies               | Hemolytic-Uremic syndrome             |
| Bartonella         | Lead                        | Hypersplenism              | Pyruvate kinase deficiencies    | Thrombocytic Thrombocytopenia Purpura |
| Babesia            | Copper                      |                            | Hemoglobinopathies              | Aortic stenosis                       |
| Clostridial sepsis | Oxidizing agents            |                            | Membrane defects                | Prosthetic valve leak                 |

Adapted from Approach to the diagnosis of hemolytic anemia in the adult. *UptoDate*, 2015.

When direct hyperbilirubinemia is present a CBC with differential, LFTs, and a prothrombin time (PT) will help identify the presence of primary hepatic failure, primary intraductal or biliary tree disease, or the presence of obstructive pathology. Painless jaundice with a posthepatic obstructive source should prompt a search for primary or metastatic malignancies of the liver, gallbladder, or pancreas. The presence of eosinophilia should raise suspicion for a parasitic process.

Viral hepatitis will typically cause direct (conjugated) hyperbilirubinemia along with transaminitis (with or without elevation of alkaline phosphatase), indicating a primarily hepatocellular process. Viral hepatitis may develop gradually, so that patients may only present when significant jaundice



ensues. If a viral etiology is suspected, serologic studies should be performed. If an autoimmune disease is suspected, relevant tests should be ordered, though this may be done as an outpatient if close follow-up is arranged.

Toxin exposure, both common and uncommon, can result in acute, chronic, or acute-on-chronic liver failure. A careful occupational history may reveal exposures to chemicals in the workplace (arsenic, carbon tetrachloride, vinyl chloride). Acutely ingested or chronically used over-the-counter medications (acetaminophen, NSAIDs), commonly prescribed medications (isoniazid, amoxicillin–clavulanic acid, TMP-SMX, valproic acid, nitrofurantoin, etc.), and complementary, alternative, or herbal medications (ackee fruit, camphor, kava leaves, etc.) can all cause hepatic failure. Subacute ingestion of plants or fungi (amanita mushrooms) can also result in hepatic failure.

There are several imaging modalities used to evaluate the liver, gallbladder, and biliary tree. Ultrasonography (US) is an excellent tool to evaluate the presence of gallbladder stones and dilation of intrahepatic ducts, but it is operator dependent and lacks the sensitivity and specificity of endoscopic retrograde cholangiopancreatography (ERCP) in evaluating extrahepatic and common biliary ductal etiologies. Nevertheless, US is inexpensive and widely available and is the initial imaging study of choice. Computed tomography (CT) does not identify gallstones as well as US or ERCP; however, it has better sensitivity and specificity for hepatic abscess, hepatic neoplasms (primary or secondary), and periampullary neoplasms and masses. CT should be considered the modality of choice when suspicion is high for malignancy in obstructive disease.

The disposition of the patient will depend on the suspected underlying pathology as well as the patient's clinical condition (vital signs, tolerance of oral intake, overall appearance, etc.). The asymptomatic patient with jaundice can be discharged safely home if acute and acute exacerbations of chronic underlying processes are excluded; prompt ambulatory follow-up for further evaluation should be arranged.

## KEY POINTS

- Painless jaundice may reflect ongoing GI bleeding. The initial assessment should identify patients at risk for variceal bleeding and ulcers.
- Hemolytic anemia is another potentially life-threatening cause. It

presents with unconjugated hyperbilirubinemia.

- When studies suggest a posthepatic disease process, the differential diagnosis should include primary or secondary malignancies as well as parasitic infectious diseases (clues: travel history or eosinophilia).
- Toxins (both common and uncommon) can produce liver failure with jaundice. Asking about acetaminophen intake; use of complementary and alternative remedies, teas, and plant-based medicines; and workplace chemical exposures is important and often ignored.

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## ERCP CAN CAUSE A LOT OF COMPLICATIONS!

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**ABRAHAM FLINDERS, MD**

Endoscopic retrograde cholangiopancreatography (ERCP) was developed in the 1960s and is a diagnostic and therapeutic procedure for biliary and pancreatic duct disease. It involves direct visualization of the stomach and duodenum via endoscope with contrast capabilities to visualize the ductal network. Instruments are used through the endoscope for biopsy, excision, stone removal, and sphincterotomy. ERCP has many well-documented complications. A survey of prospective studies that included approximately 17,000 patients reported a 6.5% incidence of complications. The most common are pancreatitis, sepsis, perforation, and hemorrhage. As the procedure has become more common, it has moved to the outpatient setting where patients are monitored for a mere 4 to 6 hours postprocedure and then deemed safe for discharge. Thus, emergency department (ED) providers must be prepared to diagnose and treat complications of ERCP, which will most frequently present with abdominal pain.

Acute pancreatitis is common in the ED and typically presents with abdominal pain, nausea, and vomiting. The incidence is approximately 3% in those undergoing ERCP. Any patient that presents with abdominal pain post ERCP should have lipase levels drawn. The manipulation from ERCP alone will elevate lipase somewhat, but usually less than three times the upper limit, and it should clear by day 2 postprocedure. ERCP-induced pancreatitis should be managed as any other case of pancreatitis but with a lower threshold to pursue imaging as it can mimic other complications such as perforation. If the patient is toxic appearing, admission (to an intensive care unit) for intravenous (IV) hydration, bowel rest, and observation is indicated, as is urgent evaluation by a general surgeon. In addition, empiric IV

antibiotics are appropriate in this setting, as is computed tomography of the abdomen and pelvis (CTAP) with IV contrast. Piperacillin-tazobactam or imipenem can be used as monotherapy or, alternatively, a fluoroquinolone combined with metronidazole.

Post-ERCP sepsis has an incidence of 0.5% to 2%. The most common etiology is acute cholangitis/cholecystitis, but patients can also present with sepsis due to liver abscess, infected pancreatic pseudocyst, viscus perforation, and pancreatitis. Patients typically present with abdominal pain and abnormal vitals including fever and shock. Suspect cholangitis when the patient has the constellation of jaundice, right upper quadrant pain, and fever. If the patient is stable, start with a right upper quadrant abdominal ultrasound (US) but have a low threshold to order a CTAP with IV contrast. These patients should be resuscitated with IV fluids, be started on broad-spectrum antibiotics, have a surgical or gastroenterology consult, and be admitted to the intensive care unit. The treatment often requires a repeat ERCP, so contacting a consulting surgeon or gastroenterologist is key.

Another important differential diagnostic consideration in the patient with post-ERCP abdominal pain is perforation. Perforation can present dramatically with an esophageal free wall rupture and tension pneumothorax or much more subtly with an asymptomatic collection of retroperitoneal free air. An upright chest radiograph with a view of the diaphragms is a good starting point if the patient presents with peritonitis, but the most sensitive imaging modality is once again CTAP with IV contrast. If perforation is suspected, patients should be made NPO and have nasogastric decompression, IV antibiotics, and surgical consultation.

When patients present with shock post ERCP, hemorrhage should be considered given the risk of bleeding from vascular injury. Most commonly, this occurs after sphincterotomy, and it is generally not excessive. Rarely, however, bleeding is severe, and it should be managed accordingly. Evaluation and management include serial hemoglobin measurements, a type and screen, and blood transfusion as needed. If the patient is stable for imaging, CTAP with IV contrast can help localize the pathology. Coagulopathy may be seen in patients with cirrhosis, and bleeding may also occur and be difficult to control in anticoagulated patients. Reversal agents should be considered in those who bleed severely and are not responsive to blood transfusion. A surgeon or gastroenterologist should be consulted from the ED since many of these bleeds are handled endoscopically or by laparotomy, but some may require interventional radiology for embolization.

## KEY POINTS

- ERCP has revolutionized the diagnosis and treatment of patients with biliary obstruction; however, it comes with a significant rate of complications.
- Acute pancreatitis, sepsis, perforation, and hemorrhage can all occur post ERCP.
- In ill-appearing patients, resuscitation with IV fluids, cross-sectional imaging (e.g., with CTAP), empiric antibiotics, and early specialist consultation are indicated.

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# **DON'T BE AFRAID TO ORDER A CT ON A PREGNANT PATIENT IF SHE REALLY NEEDS IT**

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**MARITA M. HARRIS-NADDELL, MD AND  
MICHELLE D. LALL, MD, MHS, FACEP**

Radiation exposure in pregnancy is a complex topic. Physicians remain cautious when weighing the need to image a pregnant patient versus the potential harm to an embryo or fetus. According to the American College of Radiology (ACR), the low amount of radiation in a plain film does not cause harm to a developing fetus. However, computed tomography (CT) studies can vary in levels of exposure and should be used only after appropriate risk/benefit analysis.

The ACR has released a practice parameter to guide clinicians in determining which diagnostic studies to order when imaging pregnant or potentially pregnant women with ionizing radiation. The American College of Obstetricians and Gynecologists (ACOG) has also released guidelines for imaging in pregnancy. According to ACOG, the possibility of radiation exposure to a fetus should never prevent medically indicated imaging for a pregnant patient. Both of these guidelines were most recently updated in 2014.

The ACR has summarized radiation effects based on gestational age. The risk of radiation-induced central nervous system (CNS) effects is greatest at 8 to 15 weeks of gestation. At any gestational age, radiation exposure of <50 mGy has not been shown to cause harm. Imaging that delivers 50 to 100 mGy in patients >18 weeks pregnant has also not been shown to cause harm to the developing fetus. Imaging with greater than 100 mGy may cause

detrimental effects at all gestational ages. The ACR reports that a 20-mGy dose of radiation to the fetus represents an additional lifetime cancer risk of ~0.8%. [Table 100.1](#) shows radiation doses for common imaging studies.

**TABLE 100.1 ESTIMATED FETAL EXPOSURE FROM SOME COMMON RADIOLOGIC STUDIES**

| IMAGING STUDY                       | FETAL EXPOSURE    |
|-------------------------------------|-------------------|
| Chest x-ray (two views)             | 0.0002–0.0007 mGy |
| Abdominal film (single view)        | 1 mGy             |
| Hip film (single view)              | 0.07–0.20 mGy     |
| Barium enema or small bowel series  | 20–40 mGy         |
| CT scan of head or chest            | <10 mGy           |
| CT scan of abdomen and lumbar spine | 35 mGy            |

1 mGy = 0.1 rad; amount of energy deposited per kilogram of tissue.

Adapted from American College of Obstetrics and Gynecology. ACOG Committee on Obstetric Practice. ACOG Committee Opinion. No. 299, September 2004 (replaces No. 158, September 1995). Guidelines for diagnostic imaging during pregnancy. *Obstet Gynecol.* 2004;104(3):647–651.

Ultrasound (US) and magnetic resonance (MR) are the preferred diagnostic modalities in pregnancy; however, MR is not always available. CT should not be withheld if needed to make a critical diagnosis. In addition, intravenous (IV) contrast should be ordered only if really necessary. There is some concern that it may interfere with fetal thyroid function as IV contrast does cross the placenta. Animal studies, however, have not shown teratogenic effects. According to ACOG, contrast agents are unlikely to cause harm.

Both the ACOG and the ACR recommend using imaging modalities that are not associated with ionizing radiation, such as US and MR, if at all possible. CT imaging of the abdomen and pelvis in pregnant patients is most commonly ordered in the settings of suspected appendicitis and trauma. When evaluating a pregnant patient with possible appendicitis, the most common nonobstetric surgical emergency in pregnancy, US is the preferred first-line imaging modality. If the US is nondiagnostic, MR is recommended as the next best imaging study. If MR is unavailable, CT should be performed. In a stable patient with blunt abdominal trauma, x-ray and US are performed first. However, US has diagnostic limitations in the pregnant trauma patient; CT remains the most accurate and cost-efficient tool when

evaluating blunt abdominal trauma. Single-phase CT of the abdomen and pelvis usually delivers less than 35 mGy, and with dose-sparing protocols, the radiation dose is typically 10 to 25 mGy. There is no significant evidence that a single CT of the abdomen and pelvis will be detrimental to a growing embryo or fetus. However, dose-saving or adapted protocols should be used whenever possible.

Informed consent should always be obtained, and every effort should be made to decrease the amount of radiation required for the study. According to the ACR, when obtaining patient consent, a realistic overview of the limited risk and the beneficial role of the imaging should be explained to the patient in understandable language. They state, “Conveying information in a positive, rather than negative, format is useful in helping a patient understand an accurate perspective of risk.” Informed consent should be documented in the medical record.

## KEY POINTS

- Any diagnostic imaging required to make a critical diagnosis should be ordered if the benefit to the mother outweighs the risk to the embryo or fetus.
- A single CT scan of the abdomen and pelvis is unlikely to cause fetal harm.

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## KNOW HOW TO DEAL WITH THE DISPLACED PEG TUBE

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As the number of physicians capable of placing percutaneous endoscopic gastrostomy (PEG) tubes has increased, so has the number and type of PEG tubes being placed. The PEG tube is placed for feeding access as well as for gut decompression. Nearly 10% of patients with a PEG tube suffer some sort of malfunction, with dislodgement occurring in 1.6% to 4.4% of patients. In the emergency department (ED), we need to be familiar with PEG tubes and comfortable with decision-making when these patients come to the ED complaining of tube dislodgement.

The PEG tube consists of a single-lumen tube protruding into the stomach, with a fixed internal bolster and a sliding external bolster. When normally situated, there should be a 1 to 2 cm of movement of the tube before reaching the external bolster. A gauze square is usually placed between the skin and the external bolster to collect any moisture that may gather. Normally, the skin surrounding the wound should be without erythema, exudate, or drainage. Irritation dermatitis can occur at the insertion site as well but should typically be a mild redness only. A common error is to miss an infection (redness, pain, warmth, drainage, fever) at the insertion site and to mistakenly ascribe symptoms and signs to irritation dermatitis. The two must be carefully differentiated in each patient. Abscess, wound infection, and necrotizing skin infection should be considered in patients who show systemic signs of infection, remembering that patients who are immunocompromised may not manifest all of the typical signs.

Dislodgement of a PEG tube occurs more frequently in the agitated,

demented, or delirious patient. The decision to replace the PEG tube in the ED depends on the amount of time elapsed since the PEG tube was placed, as well as the amount of time the PEG tube has been dislodged.

The PEG tube's initial placement involves controlled perforation of a hollow organ with formation of a gastrocutaneous fistula. The PEG tube holds the anterior stomach to the peritoneal wall. Eventually, the stomach becomes attached to the abdominal wall as adhesions form. The tract is considered mature in ~10 to 14 days, although the literature describes a range of 1 week to 6 months. Maturation will take longer in elderly or malnourished patients as well as those with acquired immune deficiency disorder (AIDS), cancer, or diabetes; those who have undergone radiation therapy; or those who have an otherwise compromised immune system.

When a tube becomes dislodged, history from the patient (and/or caregiver) and examination are important. It is especially important to find out the length of time that the PEG tube has been present, as well as how long it has been out. PEG tubes that become dislodged before the month-long maturation period is complete should not be replaced blindly. Lack of maturation of the tract means that there is a high probability that the stomach has fallen away from the abdominal wall. Blind placement of a PEG tube may result in the tube being placed in the peritoneum. Moreover, these patients should be considered to have a perforation, with initiation of antibiotics, nasogastric tube placement, and surgical consultation. In these cases, PEG tube replacement should be performed by a specialist using endoscopic techniques in a controlled setting.

A PEG tube that has been present for over 1 month is considered mature, and a replacement PEG tube can be replaced blindly into the fistula with minimal risk to the patient. Replacement should not be delayed, as the gastrocutaneous fistula will begin to close 4 to 48 hours after dislodgement. Be sure to inflate the interior balloon. If a replacement PEG is unavailable, a Foley catheter can be used to maintain patency of the tract and can be used for feeding purposes until a replacement PEG tube can be inserted. It should be noted that the mechanism for dislodgement, that is, trauma, or external traction, can result in disruption of a mature tract and can result in peritoneal placement if a PEG is inserted blindly. For this reason, confirmation of placement should always be performed. This can be achieved using an abdominal x-ray with radiopaque contrast injected into the tube.

Common errors in the ED management of these patients include

- 1) Failure to consult a specialist when an immature fistula track exists and proceeding with blind replacement

- 2) Failure to promptly maintain the fistula track and prevent its closure with a Foley catheter or replacement PEG tube

## KEY POINTS

- Consider infection of the tract in a patient with redness and tenderness, especially when systemic signs of infection are present.
- A confirmatory dye study after tube replacement in a patient with a mature fistula track.
- In patients with immature tracts (<4 weeks), specialist consultation should be obtained.

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# COMMON PITFALLS IN POINT OF CARE ULTRASOUND OF THE GALLBLADDER!

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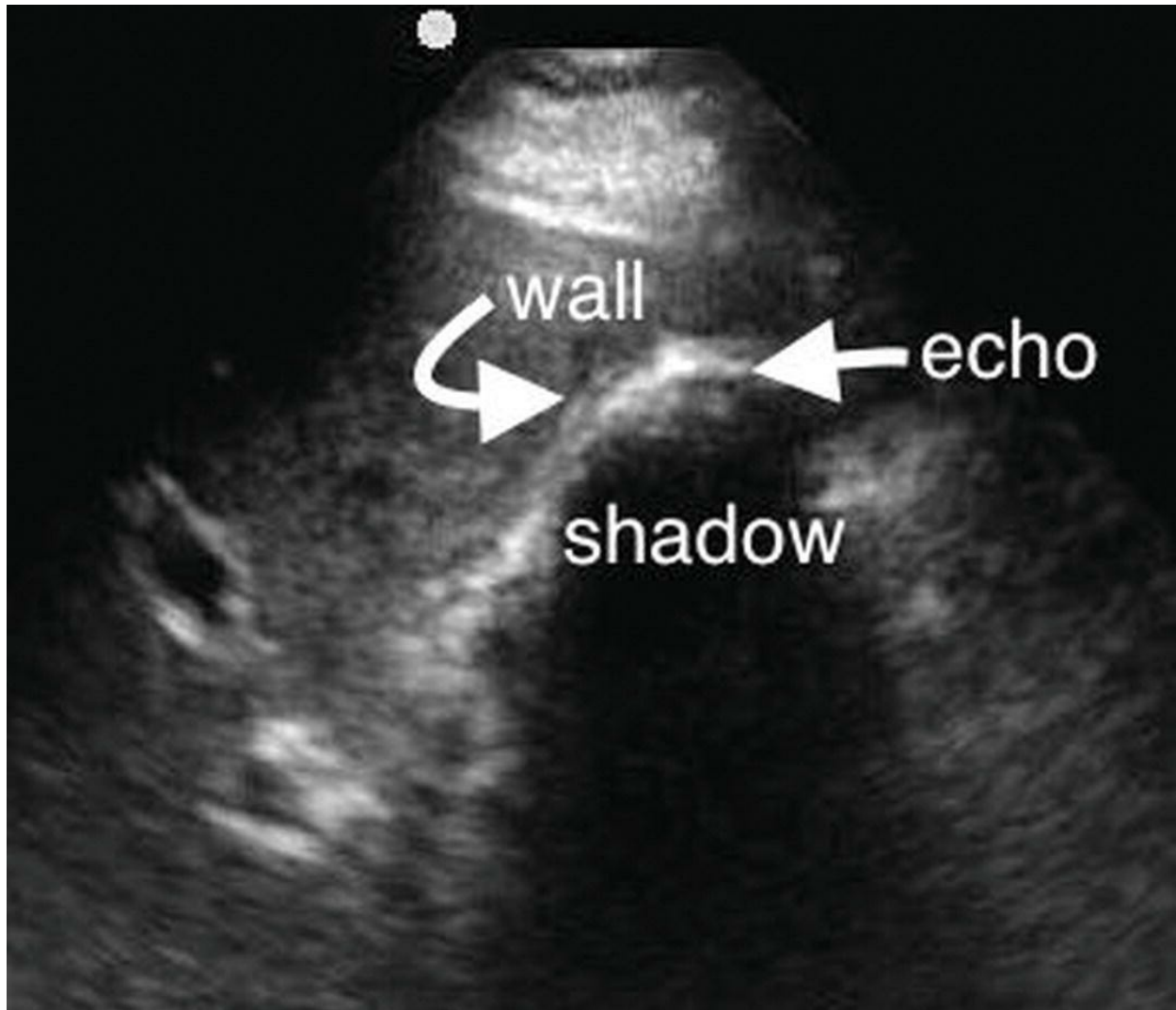
**KRISTIN BERONA, MD**

Emergency point-of-care ultrasound to evaluate for gallbladder disease has been utilized since the late 1980s. Emergency physicians are very good at detecting gallstones and cholecystitis, with reported sensitivities from 88% to 96% for gallstones and 87% for cholecystitis. However, there are specific tips and entities to be aware of to avoid missing gallstones or misinterpreting other pathologies for gallstones and cholecystitis.

A complete and full evaluation of the gallbladder is necessary to not miss stones. A full evaluation includes two views of the gallbladder in long axis and in short axis interrogating from the fundus through the neck, measurement of the anterior gallbladder wall, and measurement of the common bile duct as medial as possible. Stones are generally hyperechoic (bright), have a dense shadow, and are mobile. However, stones smaller than 4 mm may not shadow and stones impacted at the gallbladder neck may not move. The gallbladder neck is an area that calls for meticulous evaluation because gallstones there are often missed. To aid in evaluation of the gallbladder neck, you can have the patient take a deep breath, or place the patient in a left lateral decubitus position.

Another common misinterpretation is ironic: thinking that there are no gallstones when the gallbladder is so full of stones that the gallbladder itself is easy to miss, resulting in the clinician interpreting the scan as “contracted” or “indeterminate.” When the gallbladder is completely full of stones, no bile or hypoechoic area within the gallbladder is visible. This is called the “*wall*

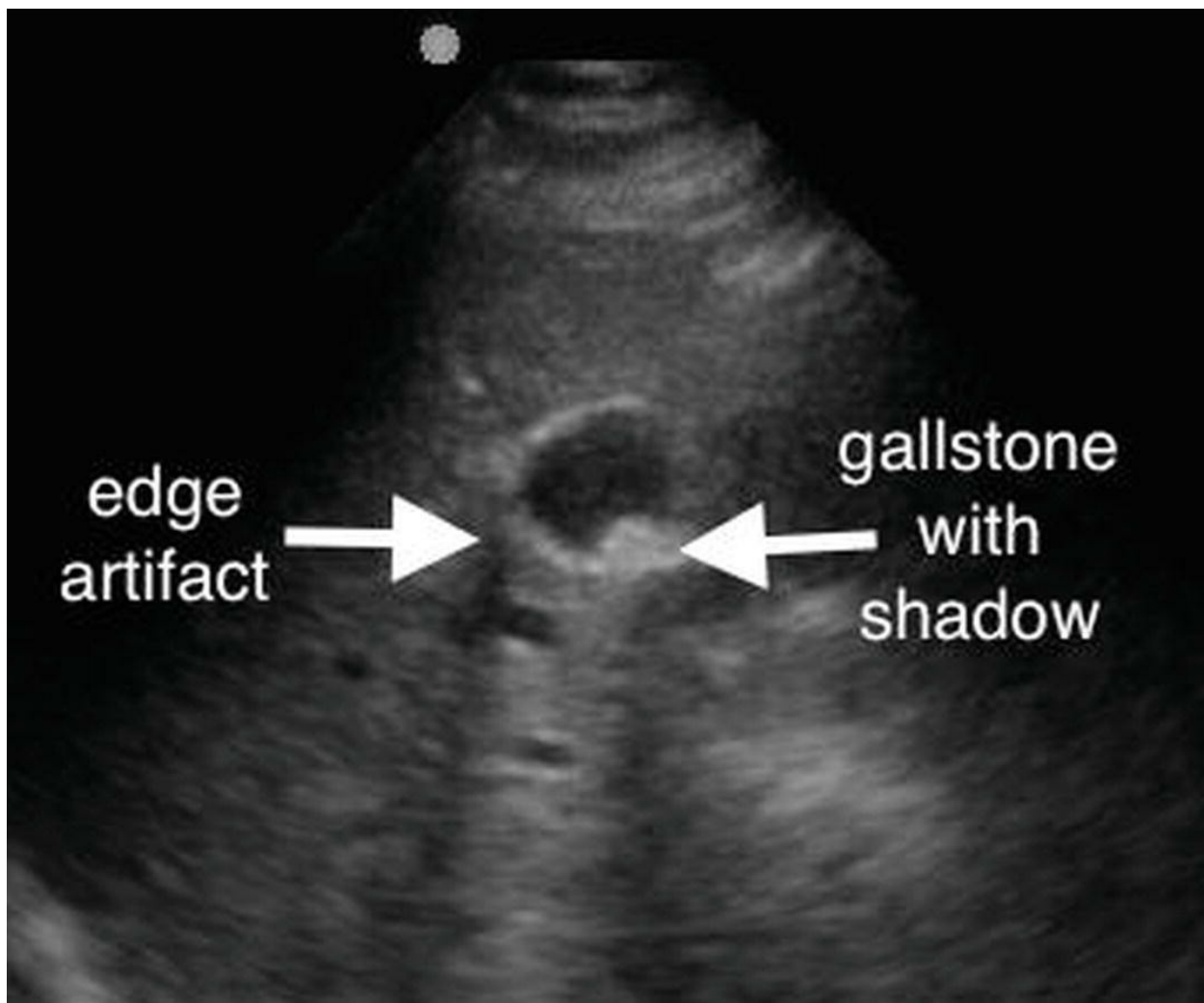
*echo shadow*” (WES) sign (see [Figure 102.1](#)). All that is seen is the hyperechoic wall, another line of echoes from gallstones, and then a dense shadow. The WES sign makes it very difficult to interpret wall thickness, but it is important not to mistake it as negative for stones.



**Figure 102.1** The wall echo shadow sign in a patient with a gallbladder full of stones.

What about the false positives? There are many instances where we might interpret the scan as positive for stones when they are not in fact present. The most common mimicker of a stone is a *gallbladder polyp*. These can be differentiated from stones in that they are not dependent or mobile and do not shadow. Gallbladder polyps need outpatient follow-up, but the patient is usually asymptomatic. *Air in the duodenum* adjacent to the gallbladder can also be misinterpreted as positive for gallstones. The air in the duodenum is hyperechoic, but does not have dense shadowing, and when the patient is placed in left lateral decubitus, it becomes clear that the

hyperechoic material (air) is outside of the gallbladder. Lastly, an ultrasound artifact called *edge artifact* can mimic a stone because it creates a shadow. Edge artifact occurs when the ultrasound beam encounters two different tissues with contrasting sound propagation or hits a curved structure. The change in beam direction causes the US beam to not be reflected back to the transducer as expected and a shadow results. These shadows occur at the edge of the gallbladder and will not have a hyperechoic stone associated. [Figure 102.2](#) demonstrates shadowing from both a stone and edge artifact.



**Figure 102.2** Shadowing from both a stone and edge artifact.

Cholecystitis is diagnosed on ultrasound when there are gallstones or sludge associated with secondary signs of infection: a positive sonographic Murphy's (maximal tenderness when ultrasound probe pushes directly over the visualized gallbladder), gallbladder wall thickening ( $>3$  mm), or pericholecystic fluid. However, there are several entities aside from cholecystitis that can cause gallbladder wall thickening, including a contracted gallbladder (i.e., after eating), ascites, pancreatitis, or alcoholic

hepatitis. In these cases, a thickened gallbladder wall needs to be taken in the clinical context and laboratory findings.

## KEY POINTS

- A thorough evaluation of the gallbladder in two planes is your best defense against misinterpretation. Look closely at the gallbladder neck!
- Be aware of entities that can be mistaken for stones: polyps, air in duodenum, and edge artifact.
- Contracted gallbladder, ascites, pancreatitis, and alcoholic hepatitis can all cause gallbladder wall thickening—don't be fooled into calling cholecystitis.

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**SECTION V**

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**CUTANEOUS**



# DON'T MISS NECROTIZING FASCIITIS!

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SHAUGHN KEATING, MD

Necrotizing fasciitis poses a particularly difficult diagnostic dilemma due to its rarity and often subtle initial presentation. The term necrotizing soft tissue infection (NTSI) is preferred as a more general descriptor of a collection of bacteriologically distinct infections that share a final common pathway: rapid necrosis of soft tissues, systemic toxicity, and high mortality if left untreated. The diagnostic conundrum lies primarily in that the symptoms of early NTSI are similar to that of cellulitis, leading to misdiagnosis, delayed treatment, and high morbidity. Successful treatment requires early recognition, maximal supportive care, and prompt surgical debridement.

NTSIs can be typed by their causal agent, and each has specific risk factors. Type I NTSIs are polymicrobial infections that more commonly affect those with impaired immune systems. They represent >80% of all NTSIs. Common comorbidities include diabetes mellitus, morbid obesity, and underlying kidney disease. Eponymous subtypes of Type I NTSIs have also been historically classified by location. Fournier gangrene was described in 1883 as *gangrene foudroyante de la verge* (“violent gangrene of the penis”), a fulminant, morbid, perineal infection. Ludwig angina is an NTSI of the deep oropharyngeal compartments and is caused by oral anaerobes.

Type II NTSIs are monomicrobial infections caused by Group A streptococci (GAS) or *Staphylococcus aureus*. They are distinguished by the virulence factors they produce. GAS M protein, protein F, streptococcal inhibitor of complement, streptolysins, hyaluronidases, streptokinase, cell envelope proteinases, and pyrogenic exotoxins and staphylococcal leukocidin, modulins, and alpha-hemolysin allow them to spread rapidly and cause toxic shock syndrome. While these infections are less common,

representing 10% to 15% of NTISs, their ability to affect healthy, immunocompetent individuals with seeming minor trauma makes them particularly dangerous.

A specific variant of monomicrobial Type II infection, clostridial myonecrosis, is alternatively classified as a Type III NTIS. Clostridia are obligate anaerobes and require deep inoculation in order to thrive. Infections occur most often with local devascularization from surgery or intravenous drug use or from complications of pregnancy such as retained products of conception. Like Type II infections, clostridia produce an array of toxins that potentiate its spread and systemic toxicity. These infections are notable for their rapid spread and production of gas within fascial planes.

Regardless of type, the key to NTIS management is early diagnosis leading to prompt debridement. Unfortunately, outward findings of erythema and edema may be minimal or absent in early NTISs because the soft tissues affected are deep to the skin. Later in the course, violaceous bullae or crepitus may appear. These findings are highly specific but insensitive and suggest that significant tissue necrosis has already occurred. Differentiating between cellulitis and early NTIS requires an understanding of the underlying pathology. NTISs impair and then destroy the underlying vasculature of the soft tissues allowing for their rapid advance. Severe pain out of proportion to exam is suggestive of this underlying tissue ischemia, similar to mesenteric ischemia or limb arterial occlusion.

Once clinical concern is raised, laboratory tests and imaging are often done, but may be of limited utility. Leukocytosis may be absent early in the patient's course, and nonspecific markers such as C-reactive protein fail to distinguish between NTIS and cellulitis. Prior work on laboratory scoring systems has failed to produce a sensitive and reliable tool to identify early NTISs. Gas along fascial planes in x-rays is pathognomonic but highly insensitive. CT scans may show diffuse inflammation, necrosis, gas, or fluid collections. In one study, when all these criteria were used, CT had a negative predictive value of 100%. However, cellulitis, myositis, and other nonnecrotizing myopathies can have similar findings, limiting specificity. MRI has been suggested as a highly sensitive modality, but is limited by the time taken to perform the study and similar issues with low specificity.

All patients with concern for NTIS should be started on maximal supportive care and antibiotics, pending definitive treatment. The robust inflammatory reaction often leads to large fluid shifts, and some patients may require 10 to 20 L of IV fluids throughout their course. Prior to identification of the causative bacteria, broad-spectrum IV antibiotics should be started with both MRSA and broad gram-negative coverage, commonly vancomycin

plus piperacillin/tazobactam. Clindamycin should also be started to suppress bacterial toxin synthesis and may also modulate endogenous cytokine production. IVIG has been investigated as an adjunctive therapy in severe disease to suppress the inflammatory response and may improve survival, but this is not yet part of standard emergency management.

Definitive diagnosis of NTSI requires surgery with direct inspection of the tissues. Delay to first debridement increases mortality up to ninefold. If high clinical concern exists, especially with late signs such as crepitus or frank necrosis, surgical consultation should occur immediately, and further diagnostic testing should not delay debridement. If the diagnosis remains unclear, the Infectious Disease Society of America guidelines recommend surgery after failure to respond to initial antibiotic therapy, defined as reduction in fever, toxicity, and lack of advancement. Frequent reassessments should be made with a low threshold to advocate for surgical management. If there is no necrosis visualized with a small exploratory incision, the procedure can be terminated with minimal risk. In an era when high-resolution imaging has allowed the negative surgical rate for many procedures to drop precipitously, there may be hesitation to perform a surgery when the diagnostic testing is equivocal. The astute emergency medicine provider may need to be an advocate for the patient given the extreme risk-benefit ratio of a negative surgery compared with letting an NTSI progress without debridement.

## KEY POINTS

- NSTIs may lack systemic toxicity or superficial skin findings early in their course. NTSIs caused by GAS can affect young, healthy hosts with minor or no apparent trauma.
- Pain out of proportion to exam differentiates NTSI from cellulitis. Crepitus, violaceous bullae, and skin sloughing are pathognomonic late findings, but have low sensitivity.
- Include clindamycin in NTSI treatment because it stops the bacterial production of locally and systemically active toxins.
- Definitive diagnosis and treatment can only be made with surgery and debridement. Mortality increases with delayed surgical intervention, so obtain surgical consultation early.
- A negative exploratory incision has low risk. Advocate for surgical exploration on all patients if there is high clinical concern or progressive infection, even if imaging findings are nonspecific.

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## **SJS AND TEN: ARE THEY DIFFERENT?**

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**ARUN NAIR, MD, MPH**

### **WHAT IS SJS/TEN?**

Recognize that these two entities are actually part of a spectrum with the same underlying pathophysiology. It is acute disseminated epidermal necrosis secondary to a hypersensitivity reaction to some nonnative agent—it is most often due to a drug, but infections are not infrequently the cause. It is the act of the epidermis separating from the dermis anywhere that is called mucocutaneous. The pathophysiology is immune modulated and multifactorial and super interesting, but you only need to know it in broad strokes to provide excellent care. This is a burn, both on the outside and inside, but with no associated thermal injury. The severity of the burn is based on the body surface area (BSA) involved just like any other burn but with one major caveat. There is often involvement of the unseen mucocutaneous tissues—the alimentary, pulmonary, and genitourinary tracts—which are not accounted for in traditional BSA calculations. Burnt skin does not perform its usual functions, and it scars. The burn keeps growing if the offending agent is still present. If you keep this overarching understanding in mind, then the disease process and its treatment make sense.

### **SKIN FINDINGS**

Armed with the understanding that the epidermis is separating from the dermis en masse, the skin findings including the Nikolsky sign should make more sense. The epidermis is cleaving off the dermis as an intact layer or

sheet and can be seen when a shear force is applied to the skin. Another way this can be tested is with already formed blisters— if the entire blister can be moved by applying gentle pressure to one side, it demonstrates that the epidermal/dermal interface has been disrupted. This sign is nonspecific and can be seen with other conditions, but its presence combined with any mucosal lesion (most often seen on lips or in the mouth, but also remember the conjunctiva and cornea) should cause serious consideration of a diagnosis of the spectrum of SJS/TEN. Lesions can range from the pathognomonic flat targetoid red and white lesion (not to be confused with the raised/edematous blue, white, and red lesions of erythema multiforme target), which may become confluent, develop into thin-walled blisters, or necrose into erosions. As the disease process continues, sloughing off of large areas of skin can be seen on palms, soles, tongue, etc. Except when the areas become superinfected, there should be no significant edema or induration noted, and the skin sheets should be thin and seem “topical.”

## **LOSS OF BARRIER**

The epidermis’ job is to separate one’s insides from the outside world. It is the first layer of protecting homeostasis, keeping our fluids and heat within and preventing pathogens from without. Treatment of the skin findings in SJS/TEN is the same as any other skin burn: provide analgesia, prevent infection, and replace fluids and electrolytes as needed. Since there is no thermal injury, the inflammatory response is not as exaggerated, and there are less insensible losses. Replacing fluid at the rates recommended by the Parkland formula will likely overestimate fluid deficit causing “overresuscitation” and its sequelae ranging from mild peripheral edema to compartment syndrome and acute pulmonary edema. However, the patient may require a large initial bolus to make up for fluid deficits depending on time of presentation. Consider early ultrasound examination of IVC collapsibility for estimating initial fluid status and placement of a Foley for continuous urine output monitoring with a goal of 0.5 to 1 mL/kg/h.

As the inciting agent is often a drug, DO NOT dress the skin with Silvadene (sulfa derivative) unless directed to do so by a burn specialist and only if the causal agent has been definitively identified. The lesions should be kept moist, protected, and sterile with nonadherent petroleum gauze. A large percentage of patients with significant BSA involvement will go on to develop skin infections. Try to prevent this as much as possible to avoid the risk of adding another drug. Prescribing an antibiotic to someone undergoing a hypersensitivity reaction is a risky option at best.

With the loss of the epidermis, the patient has lost his or her primary

means of thermoregulation. Keep your patient as minimally exposed and for the shortest time possible. Your patient may require warmed fluids or an external heating device such as a Bair Hugger to maintain their temperature.

## RECOGNIZE INTERNAL INVOLVEMENT

Our mucosal tissue allows for the diffusion of gases integral in ventilation and oxygenation as well as the ciliary action responsible for mucus clearing in our airways. Patients may progress to respiratory compromise from pulmonary edema or obstruction and may require mechanical ventilation. In this situation, care must be taken during intubation to not cause further airway compromise by desquamating the tongue or other structures down into the airway. The alimentary epithelia allow transport of everything from ions to macromolecules and are integral in absorbing and retaining free water—patients with significant GI involvement may require parenteral nutrition. Our specialized corneal epithelia provide the transparency required for proper vision. The fibrinous exudates caused by the loss of the mucosal layer can cause scarring and stricture in luminal structures and can lead to permanent loss of vision through corneal scarring. Early recognition and management of these conditions are essential.

## DISPOSITION

Don't get too caught up in measuring BSA. If there's minimal BSA involvement, the patient looks good, and the causal agent is identified and ceased, then the patient's prognosis can be great and may get away with hospital observation. If on the other hand, the size of lesions combined seems bigger than the front of the torso, its  $>10\%$ , and you should be calling your local burn center ASAP for transfer. If its  $<10\%$  but they look sick, better to err on the side of transferring. Bad thermal or chemical burns go to the burn center—the same applies here. Greater than  $10\%$  BSA means the patient is in the overlap SJS/TEN phase and mortality  $>5\%$ . If your patient is on this part of the spectrum of SJS/TEN, the patient goes to the burn center. This is not something for a novice practitioner to manage nor is this the patient that can be admitted so that dermatology can see him or her once the weekend is over. This level of mortality automatically necessitates ICU level of care and requires the expertise that can only be delivered at a burn center. See [Chapter 105](#).

Most importantly, try to identify the causal agent and cease exposure. The disease process will continue to worsen as long as there is ongoing exposure to the agent—stop all medications possible! The most important

predictors of outcome are how early the exposure is stopped and how quickly the patient is transferred to a burn center.

## KEY POINTS

- SJS/TEN is a spectrum of a hypersensitivity reaction to an offending agent in which the mucocutaneous epidermis is separating from the dermis en masse.
- Don't forget that the burn is also on the inside with consequences for the GI, GU, and pulmonary systems.
- Treat SJS/TEN the same as you would any burn - maintain normothermia, replace fluid losses, and protect affected areas from infection.
- The most important predictors of patient outcomes are how early the offending agent is stopped and how quickly they arrive at a burn center - do not be the cause of the delay!

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# THE SPECTRUM OF TEN

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**ALEXANDER JENSON, MD, MPH**

Toxic epidermal necrolysis (TEN) is a severe form of an adverse autoimmune reaction (usually drug induced) that involves keratinocyte death and separation of the epidermis from the dermis of skin and mucous membranes. It is part of a spectrum of severe epidermolytic reactions that include Stevens-Johnson syndrome (SJS). Although rare, it still affects 2 per million per year and has an overall mortality rate of 30%. In an emergency department, rapid recognition (1), determination of severity (2), early initiation of supportive care (3), and monitoring for life-threatening sequelae (4) are essential.

## RECOGNITION OF TEN

Although characterized by cutaneous and mucosal involvement, TEN is frequently preceded by a prodrome consistent with a viral illness (cough, fever, congestion, malaise). Typically, a few days after the prodrome, painful erythematous macules develop symmetrically over the trunk, face, palms, and soles. Over 90% of patients with TEN will have mucosal involvement, including buccal, genital, and ocular (conjunctival or corneal) erythema and erosions, which can cause dysphagia, visual changes, and pain. In the second phase, these patches develop into bullae and detach. At presentation, skin can vary from macules to blisters/bullae to erosions.

A history of present illness (HPI) must include discussion of recent medications, as 80% to 95% of TEN cases develop as result of a drug reaction, but the absence of this history should not prevent diagnosis as identification of the offending drug often occurs post hoc. Drugs that can commonly induce SJS/TEN can be divided simply into categories and include antiepileptic drugs (carbamazepine, phenytoin, and phenobarbital),

sulfonamides (notably Trimethoprim/Sulfamethoxazole [TMP/SMX]), and antibiotics (including penicillins, cephalosporins, carbapenems, and quinolones). Usually, the reaction begins between 1 and 8 weeks after initiation of the agent, and for chronic medications, TEN risk drops precipitously after 8 weeks. Other causes (although rare) for TEN include infections such as *Mycoplasma pneumoniae*, cytomegalovirus, dengue fever, a paraneoplastic reaction, immunizations, and even reactions to contrast medium. Cohort studies have shown that patients with malignancy, HIV, lupus, and collagen vascular disease are at higher risk of developing TEN when exposed to these medications.

On exam, the hallmark rash is present over mucosal surfaces (lips, oropharynx, genitalia) and is notable for patches peripherally with confluence over face, palms, soles, and trunk. The rash is painful and progresses to blistering and epidermal sloughing, which can be precipitated by lateral pressure (Nikolsky sign). The percentage of body area defines TEN, with <10% body surface area (BSA) characteristic of SJS, 10% to 30% of TEN/SJS overlap, and >30% for TEN.

Importantly, the differential for TEN includes IgA dermatosis, paraneoplastic pemphigoid, and staphylococcal scalded skin syndrome, which can all present with Nikolsky sign and bullae. Additionally, an important distinction is made with drug eruption with eosinophilia and systemic symptoms (DRESS), which has bullae and lip erosions, but without epidermal sloughing and histologically distinct. Additionally, erythema multiforme may resemble the early painful patches of TEN, although epidermal death and bullae are infrequent.

## SEVERITY OF TEN: SCORTEN

Given the high mortality rate of TEN, a rapid assessment of prognosis is essential for prioritizing resources. As a result, the SCORTEN system has been developed and validated repeatedly to guide prognosis, management, and disposition for patients presenting with TEN.

The components of SCORTEN are age (>40 years), malignancy, tachycardia (>120 bpm), percent of BSA (>10%) detached, serum blood urea nitrogen (BUN) (>28 mg/dL), serum glucose (>252 mg/dL), and serum bicarbonate (<20 mEq/L). Each is a binary variable that adds one point to the score, up to 5 points. Each point adds to the relative risk of mortality during hospitalization, from 3% (0 to 1 point) to 90% (5 points). The tool has been validated at multiple burn units in the United States and abroad.

What does this mean for the Emergency Physician? Classic teaching is

that a score of two or higher in SCORTEN should prompt ICU level care at a burn center. However, it is important to remember that this is a progressive process, and the patient's initial presentation may be anywhere in the disease course. If there is real concern for TEN, the patient needs a level of care that can only be delivered at a burn ICU. Obviously, this decision should be made in discussion with the local burn center and should also take into account body surface involved, fluid repletion requirements, and other systemic illness that may complicate care.

## TREATMENT GUIDELINES FOR TEN: WHO, WHAT, WHERE, AND WHY?

Before a discussion of who should be involved in the management of a patient with TEN, it is crucial to stop the offending agent. In an ED setting, this means both a good medication reconciliation and discussion with dermatology. Unless absolutely necessary, cease all home medications if TEN is suspected given the concern for complications.

Next, the **who**. This patient **must** be transferred to the local burn center for management. Only a burn center has the combination of ICU experience and large wound management. Multiple studies have shown that delayed transfer to burn facilities directly increases mortality, so this patient should be transferred as soon as reasonably possible.

Then, the **where**. Many TEN patients, especially those with other critical illness, will require ICU level care, as discussed above. When discussing the case with your local burn center, have a low threshold to advocate for a high level of care for these patients, given complex fluid management and unseen visceral organ involvement.

While disposition is important, **what** should be done in the ED prior to transfer? Discuss with your local burn specialist regarding care, but there are standards for management. First, fluids are a standard requirement for burns and similarly for TEN/SJS. TEN differs from classic burns in two ways. First, there is less microvascular injury, and cytokine response is less than in a burn, decreasing the relative amount of insensible losses. Also, there is heightened concern for pulmonary involvement in TEN, increasing potential morbidity of overresuscitation and pulmonary edema. Nevertheless, these patients may require up to 5 to 7 L/24 h of fluids. This should be titrated to urine output of 0.5 to 1 mL/kg/h, as with other resuscitation. Given concern for ureteral injury and close monitoring of output, a Foley catheter should be placed.

As with burns, bacterial superinfection is a common complication, and ED care should start infection prevention. While transfer should not be delayed for wound care, wounds should be cleaned if possible and should be lightly covered with nonocclusive dressings as is standard in burn wound management. Do **NOT** use sulfa-containing medications (Silvadene included) as burn covering dressing prior to transfer, as this could have been a cause of the TEN. Try to minimize dressing changes to reduce sloughing of skin from contact. All intravenous lines should be placed as far away from burned skin as possible.

In addition to supportive care (fluids, pain relief), TEN's hallmark mucosal involvement creates extradermal manifestations that the emergency physician must be aware of and potentially manage early in this patient's care. Up to 25% of patients may have pulmonary involvement, including bronchiectasis, bronchiolitis obliterans, and acute respiratory distress syndrome (ARDS). Patients may frequently present with hypoxemia, but with a normal initial chest radiograph (sloughing is typically not visible on initial radiograph). Intubation may be complicated by oral and mucosal lesions, and special care should be taken during laryngoscopy.

In addition to pulmonary complications, gastrointestinal and ocular complications are common in TEN. These will both likely be managed at the local burn center and are not a core component of ED management of TEN. Prior to transfer, patients should be NPO, and ocular symptoms can be managed with lubricating eye drops and erythromycin ointment. Ophthalmology should be involved early to assess ocular manifestations and lyse ocular adhesions but should not delay transfer to definitive care and management.

Systemic treatments for disease modification (such as corticosteroids or intravenous immunoglobulin) are controversial in TEN, with different data showing minimal to no effect on prognosis or morbidity. Any initiation of such therapy should be done in consultation with dermatology and/or the local burn center, particularly in patients with underlying diseases that may influence the pathogenesis of TEN.

TEN is a potentially life-threatening condition that must be recognized, referred, and managed by every emergency clinician. Early initiation of supportive care and transfer to the appropriate level of care is essential to reduce mortality.

## KEY POINTS

- TEN is an epidermolytic autoimmune reaction of the skin and mucous membranes. It is pathophysiologically the same as SJS, but defined as >30% BSA involvement.
- The SCORTEN system for assessing TEN prognosis includes age, malignancy, glucose, BUN, %BSA detached, and bicarbonate. A score of 3 or higher prompts ICU admission.
- Although similar to burns with the requirement for early aggressive rehydration, less fluids are required in TEN, and careful monitoring of pulmonary complications of overhydration is essential.
- Pulmonary complications, including ARDS, are common in TEN. In addition, gastrointestinal and ocular manifestations are common.
- All patients with suspected SJS/TEN should be transferred as soon as possible to a burn center. Basic wound care and fluid rehydration should be initiated by the emergency physician prior to transfer.

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## MIMICS IN CELLULITIS

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SHABANA WALIA, MD

There are many dermatologic conditions that can be strikingly similar to cellulitis, especially on first presentation to the emergency department. While some diagnoses are more chronic and indolent, others are life threatening and crucial for the emergency physician to diagnose. Distinguishing between cellulitis and other skin conditions can lead to a decrease in antibiotic use, a decrease in the development of antibiotic resistance, as well as delays in treatment of alternate and deadly diagnoses.

Cellulitis is an acute bacterial infection causing inflammation of the epidermis, dermis, and underlying subcutaneous tissue. Group A-beta hemolytic *Streptococcus* and *Staphylococcus aureus* are the most common bacteria causing cellulitis. However, in children, and less commonly in adults who are not responding to initial treatment, one must think of other causes of cutaneous cellulitis. *Haemophilus influenza* type B is a severe form of cellulitis that is accompanied by a respiratory infection. *H. influenza* cellulitis can be differentiated from more common forms of cellulitis by physical exam as the rash can have a characteristic blue-red-purple appearance. Other uncommon forms of cellulitis are *Vibrio vulnificus* and *Aeromonas hydrophila*, both of which are water-related organisms. *A. hydrophila* should be suspected if there is a history of exposure to fresh water, if treatment for streptococcal cellulitis fails, or if there are bullae and abscesses with foul-smelling exudates on physical exam. *V. vulnificus* should be suspected if there was an exposure to salt water, along with physical exam findings of large bullae and vesicles. In more aggressive and serious stages, *V. vulnificus* can progress to myositis and present similar to gas gangrene.

Erysipelas is a type of superficial cellulitis that involves the epidermis, upper dermal layer, and the superficial lymphatic channels. On history, the patient may describe a more rapidly progressing infection than cellulitis.

Similar to cellulitis, the infected area of skin can be erythematous, warm, and tender. In contrast, erysipelas can be differentiated by the raised margins and sharply demarcated edges due to the superficial nature of the infection versus the indistinct margins of cellulitis. Since Group A *Streptococcus* is the most common cause of erysipelas, the treatment and clinical management are often the same as for deeper forms of cellulitis.

Stasis dermatitis, commonly known as “Varicose eczema,” is often misdiagnosed as cellulitis in the emergency setting. It is a complication of long-standing chronic venous stasis, which is commonly a result of age-related valvular insufficiency and less commonly surgery, previous DVTs, and traumatic injury. Venous insufficiency leads to edema and extravasation of blood cells, which can result in decreased blood flow to the tissues. Patients with this condition will often have nontender, swollen, erythematous legs with areas of hyperpigmentation and scaling ongoing for several months to years. Stasis dermatitis can be secondarily infected with a superimposed cellulitis or ulcers.

Lipodermatosclerosis or “sclerosing panniculitis” can be a complication of long-standing chronic venous insufficiency and stasis dermatitis. The proposed pathophysiology is similar to stasis dermatitis resulting in decreased tissue perfusion, with the addition of further endothelial damage. This damage causes microvascular thrombi formation, which results in tissue infarction and the formation of fibroblasts and granulation tissue. On physical exam, you should notice tapering of the lower third of the legs resembling the upside-down “champagne bottle appearance,” as this disease typically affects the bottom third of the lower legs. This finding may be the only differentiating factor from cellulitis in the acute phase as patients can also develop severe pain, warmth, and redness with indistinct margins of the skin similar to cellulitis. The chronic phase is characterized by erythematous indurated skin with browning discoloration and sclerotic plaques, thus more easily differentiated from cellulitis.

Contact dermatitis is a skin reaction from an allergen or irritant that results in skin inflammation. Differentiating cellulitis from dermatitis can be simplified with a clear history from the patient. The presence of erythema or a rash at the site of an allergen exposure is a clue to diagnosing this condition. Patients may also complain of intense pruritus with the rash or have a history of allergies. Physicians should inquire about the use of new soaps, detergents, or topical creams. Contact dermatitis is a Type IV hypersensitivity reaction and thus usually occurs 1 to 2 days after the exposure. The condition improves with avoiding the offending allergen, antihistamines and mild steroid cream. Antibiotics are not a mainstay of treating contact dermatitis. Papular urticaria is another common

hypersensitivity reaction that can occur after an insect bite. It consists of pruritic papules surrounded by wheels or erythematous bases that can progress to blisters and ulcers and tends to be localized near the insect bite.

The deadliest “can’t miss” mimic of cellulitis is necrotizing fasciitis. Early on in the course of deep soft tissue infections, it can be extremely difficult to detect differences on physical exam as these infectious can share many characteristics with cellulitis such as erythema, warmth, localized swelling, and tenderness. Necrotizing fasciitis involves the deep subcutaneous tissues and spreads rapidly through the fascia and later can involve the muscle. Though the infection is usually mixed, a wide range of bacteria including gram-negative, gram-positive, and anaerobic bacteria have been implicated. Pain out of proportion to the exam should always alert the physician that a deeper soft tissue infection may be occurring. The progression of the disease is much faster than other dermatologic conditions. Within hours, the skin layers can become erythematous, swollen, and crepitant and form abscesses. Gas can be seen on radiographs, but imaging should never delay diagnosis. Early antibiotics, including broad and anaerobic coverage, and most importantly early surgical debridement are the treatment however, diagnosis is confirmed in the operating room by direct visualization of the necrotic tissue.

These conditions are common mimics of cellulitis, though not an all-inclusive list. Other physical presentations such as DVT, thromboembolism, vasculitis, viral and drug-related exanthems, fungal infections, or malignancy must be considered in the appropriate clinical setting. Most importantly, the patient should be instructed to follow up with a primary physician within 24 to 72 hours of initial presentation of the acute rash. Dermatologic conditions in immunosuppressed individuals or those not responsive to initial treatment or recurrent/chronic rashes should prompt admission or urgent dermatology follow-up.

## KEY POINTS

- Cellulitis most often presents unilaterally.
- Stasis dermatitis, the most common mimic of cellulitis, results from a long-standing history of chronic venous stasis and decreased tissue perfusion.
- Pain out of proportion to exam should prompt the emergency physician to consider necrotizing soft tissue infections.
- Observation and serial exams will aid in treatment and evaluation for



alternate diagnoses.

- A thorough history and physical exam will most often direct the clinician in differentiating cellulitis from its mimics.

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## CHICKENPOX AND SHINGLES: MORE THAN JUST A RASH

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Chickenpox is a common and usually benign childhood illness caused by the varicella-zoster virus. It is most often characterized and diagnosed by its distinct pruritic vesicular rash in various stages across the body. However, we must be cognizant of complications beyond the itchy rash and prepared to treat what may result. The most common complication of chickenpox is secondary superficial cutaneous bacterial infections. Infection is usually due to *Staphylococcus aureus* or *Streptococcus pyogenes*. Localized cellulitis may be treated with antibacterial agents. More severe infections such as toxic shock syndrome and varicella gangrenosa have also been noted. Because some studies have demonstrated a possible relationship between varicella gangrenosa and NSAIDs, it is recommended that we avoid NSAIDs in the treatment of fever and pain accompanied by chickenpox. In immunocompromised children, bullous and hemorrhagic varicellas have been seen and may be associated with thrombocytopenia or disseminated intravascular coagulation.

Beyond the skin, chickenpox has also been found to cause pneumonitis in immunocompetent populations and even more so in the immunocompromised. Typically, pneumonitis is visible on chest radiographs as diffuse interstitial nodular infiltrates. We must be suspicious of this complication in those who present with dyspnea and cough in the context of varicella rash. These cases should be treated aggressively with antiviral medication and close observation, as there may be significant mortality in the absence of treatment.

Another important complication of varicella infection is neurologic disease. These may include cerebellar ataxia, encephalitis, transverse

myelitis, meningitis, and Guillain-Barre syndrome. When the rash is noted along with neurologic findings on exam, it is important to consider such complications. The role of antiviral medications is unclear in varicella-associated neurologic diseases. However, due to limited risks of treatment, therapy is typically pursued in the case of viral encephalitis or severe disease.

After initial infection with varicella-zoster, the virus remains latent in dorsal root ganglia and sometimes reactivates in the form of shingles, or herpes zoster, as cell immunity decreases. Most cases of shingles occur after the age of 50, and risk increases with age. Shingles typically presents as a painful vesicular rash confined to one or two dermatomes, usually preceded by 1 to 5 days of skin discomfort. Unlike chickenpox, treatment is recommended for most cases as it has been shown to reduce symptoms, severity, and complications. Valacyclovir and famciclovir are the antiviral agents of choice, preferred over acyclovir. Typically, adjuvant corticosteroids may be used in those without contraindications as they have been shown to improve outcomes. It is also imperative to provide pain control to those with shingles as this pain may be very severe.

The most common complication of shingles is postherpetic neuralgia. The frequency increases with age, and it is diagnosed as pain persisting more than 30 days after the onset of zoster rash. While not a dangerous complication, the pain should not be underestimated and should be treated aggressively. Data have shown that opioids, tricyclic antidepressants, and gabapentin may be useful for treatment of pain.

In patients with zoster affecting the first division of the trigeminal nerve, perform a detailed eye exam to evaluate for the presence of herpes zoster ophthalmicus (HZO). Studies have shown that patients with eye redness in the context of herpes zoster have high likelihood of having moderate to severe disease. Other key features include photophobia, and rash in the supratrochlear division, or Hutchinson sign (erythematous skin lesions on the tip, side, or root of the nose). Not only do these patients require prompt antiviral treatment, they should also have urgent ophthalmologic evaluation as ocular diseases such as uveitis, keratitis, retinitis, and optic neuritis may pose a threat to their vision. Another visual complication of HZO or remote zoster in AIDS patients is acute retinal necrosis (ARN). This retinal injury is caused by hematogenous spread. Although the disease is slowly progressive in immunocompetent patients, in those with AIDS, it is very rapid and progresses from ARN to retinal detachment and blindness, which may spread bilaterally without treatment.

Important neurologic complications of shingles include contralateral hemiparesis, encephalitis, and other nerve palsies. Contralateral hemiparesis

may occur weeks to months after the development of rash. It is believed that zoster reactivation in the trigeminal nerve is able to spread to the cerebral arteries causing inflammation and ischemia causing contralateral hemiparesis. Patients should be treated with corticosteroids and antivirals. Of note, the infarction is irreversible despite treatment. Encephalitis associated with zoster is one of the most dangerous complications. It typically presents with fever, headache, and other neurologic findings in the context of recent zoster infection. It is usually seen in patients with AIDS and despite treatment often progresses to death. However, some reports note some benefit with high-dose intravenous acyclovir.

## KEY POINTS

- Most patients with chickenpox will not require treatment. In contrast, most patients who present with shingles within 72 hours of rash development should be treated with valacyclovir or famciclovir.
- In patients with varicella or zoster and respiratory complications, evaluate for the presence of pneumonitis, and if diagnosed, treat aggressively.
- In patients with recent or ongoing varicella or zoster and neurologic complaints, one should have high suspicion for encephalitis and contralateral hemiparesis as these have potential for high morbidity and mortality and should be treated aggressively.
- Postherpetic neuralgia may be extremely painful, and adequate pain management should be prescribed as necessary.
- All patients with HZO and eye redness should receive urgent ophthalmology evaluation.

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# **ERYTHEMA NODOSUM, NODULES, AND HYPERSENSITIVITY**

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Erythema nodosum (EN) is a delayed hypersensitivity reaction characterized by tender subcutaneous nodules typically found in a bilateral, pretibial distribution. EN rarely appears in emergency medicine literature; however, it may herald serious underlying illness that would otherwise go unrecognized. This chapter discusses management pearls for your next patient who presents to the emergency department with EN.

EN has been shown to occur from a wide variety of exposures, although up to 60% of cases are idiopathic.<sup>1</sup> The annual incidence of EN ranges from 1 to 5 per 100,000,<sup>2</sup> with women of reproductive age at higher risk.<sup>3–5</sup> EN may be preceded by a prodrome of 1 to 3 weeks that includes fever (60%), malaise (67%), arthralgias (64%), arthritis (31%), and upper respiratory symptoms.<sup>3</sup> The arthralgias may persist up to 2 years after resolution of the other symptoms.<sup>2</sup> Less frequently noted systemic symptoms include lymphadenopathy, hepatomegaly, splenomegaly, and pruritus.<sup>4</sup> The lesions themselves are typically found on extensor surfaces and are red, raised, nonulcerative, and tender. They are a few centimeters in diameter and as they heal take on a bruise-like appearance, moving from red to yellow, and finally purplish.<sup>1,4</sup>

Your principal concerns when presented with a patient with EN are to screen for dangerous causes and mimics. Leading causes are idiopathic (55%), streptococcal infections (28% to 48%), sarcoidosis (11% to 25%), drug reaction (3% to 10%), pregnancy (2% to 5%), and inflammatory bowel disease (1% to 4%). Streptococcal infection is the leading cause in pediatric populations. Rarer causes include lymphoma/leukemia, tuberculosis, HIV,

HSV, viral hepatitis, histoplasmosis, and coccidioidomycosis.<sup>1,2,4,6</sup>

Other dangerous dermatologic conditions may present similarly in their early stages. This differential includes cellulitis, erythema multiforme, envenomated spider bites, toxic epidermal necrolysis, toxic shock syndrome, bullous disease, Rocky Mountain spotted fever, and meningococcemia. Other common mimics of less consequence to emergency management include cutaneous vasculitis, nodular vasculitis, and superficial thrombophlebitis.<sup>1,7,8</sup> Atypical presentations for EN are most commonly misdiagnosed as cellulitis, trauma, or sarcoma.<sup>7,9,10</sup>

## **APPROACH TO THE PATIENT WITH ERYTHEMA NODOSUM**

Once you have ruled out other dangerous conditions, your initial screening should assess for the most common causes of EN. All patients should receive a chest x-ray to assess for sarcoidosis, tuberculosis, or other infections of the lung. You may consider testing for tuberculosis exposure with a PPD. Females of reproductive age should be tested for pregnancy. Ill-appearing patients or patients with a history concerning for cancer should receive basic blood work, with consideration of screening for HIV and viral hepatitis status, as well as blood cultures for sicker patients.<sup>6</sup>

Patients with gastrointestinal symptoms may require diagnostics or imaging in the emergency department or may simply be referred for outpatient GI workup depending upon acuity of the presentation.

It is commonly recommended to screen for recent or active streptococcal infection using throat cultures, rapid antigen test, or antistreptolysin-O antibody titer, particularly in the patient with recent symptoms of pharyngitis. However, because EN represents a late hypersensitivity process and not active infection, this is unlikely to change your acute management and should be balanced with available emergency department resources.<sup>2,4</sup>

If the workup does not identify a cause, you can provide reassurance to the patient and referral to primary care follow-up. EN typically resolves without treatment. Compression bandages and elevation of affected extremities may help provide symptomatic relief. NSAIDs (typically indomethacin or naproxen), as well as colchicine (2 mg for 3 days, 1 mg daily for 2 to 4 weeks), may provide symptomatic relief. Potassium iodide (400 to 900 mg/day) may be of benefit but is not without potential side effects, including abdominal pain, nausea, vomiting, diarrhea, and swelling. A short course of systemic steroids or injection of intralesional steroids may

provide limited benefit and but should be weighed against risks.<sup>11</sup> Other treatments inappropriate for the emergency department setting include dapsone, methotrexate, and anti-TNF agents.<sup>2</sup> If diagnostic confirmation of EN is desired, refer to dermatology for a biopsy of the lesion.

## KEY POINTS

- EN is usually idiopathic, but the most common identified causes are strep infection, sarcoidosis, pulmonary infection, systemic viral infections, pregnancy, and drug reactions.
- Beware of other dangerous rashes that may mimic EN.
- Recommended emergency department diagnostics include the following:
  - Most patients: chest x-ray, basic blood work, pregnancy test, consider strep screening
  - GI complaints: imaging, stool studies, referral
  - Sick patients: blood cultures, HIV screening, viral hepatitis screening
- Most patients can be reassured that this will resolve on its own and be safely discharged to primary care follow-up.
- The safest and simplest option for symptomatic management is a course of NSAIDs.

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# **CLASSIC IS NOT ALWAYS CLASSIC: CLASSIC RASHES**

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**DEBRA RAVERT, MD**

Rashes can be intimidating. Many are nonspecific and innocuous, but some are associated with significant pathology and cannot be overlooked. Rashes are often associated with viruses and bacteria but can also be caused by fungi, parasites, malignant processes, medications, and other chemicals. As such, a thorough history must be taken that includes questions about risk factors for immunosuppression, medication changes, allergies, and exposures. Though considered “classic,” some of the rashes in this chapter are rarely seen in clinical practice and warrant a review.

## **CLASSIC PEDIATRIC RASHES**

Measles (rubeola)—historically known as first disease, caused by the measles virus. The incidence of measles worldwide has declined sharply since the advent of effective vaccination, and the WHO (World Health Organization) has set a goal for elimination of measles in five of their six regions by 2020. Measles is highly contagious, and populations need a 95% immunization rate to control it. Recent outbreaks in underimmunized parts of the United States underscore the need for practitioners to recognize the signs and symptoms. The measles rash begins after several days of high fever, cough, coryza, and conjunctivitis (the “three C’s”) and generally starts on the face and spreads down the body, progressing from discrete macules to a confluent rash (see [Figure 109.1](#)).



**Figure 109.1 Classic measles rash.**

Scarletina/scarlet fever—historically known as second disease, most often caused by group A beta-hemolytic. Scarlet fever is generally preceded by strep pharyngitis, with associated fever, sore throat, and headache. The rash is distinguished by its “sandpaper” texture and on dark skin may be more palpable than visible. Confirmation is with strep culture from oropharynx, and first-line treatment is penicillin.

Rubella—historically known as third disease, caused by the rubella virus. The rash of rubella is similar to the measles rash and similarly begins on the face and spreads down the body. Associated symptoms are generally less severe and include low-grade fever and malaise and patients are usually nontoxic. Significant tender lymphadenopathy is classic for rubella. Rubella is uncommon but important to recognize because of its teratogenicity.

Erythema infectiosum—still colloquially called fifth disease, caused by parvovirus B19. The classic “slapped cheek” rash of fifth disease usually follows several days after a mild prodrome of fever and rhinorrhea. The rash is more common in children than adults, and associated polyarthralgia is more common in adults than children. In a significant percentage of patients, the facial rash is followed by a more diffuse, lacy rash over trunk, back, and extremities. Fifth disease is mild and self limited in most patients but is associated with anemia in sickle cell patients and immunosuppressed hosts.

Roseola—historically called sixth disease or exanthem subitum, caused by human herpesvirus 6 and 7. Most common in very young children, the roseola rash typically appears after a high fever as the child defervesces. Patients usually have few associated symptoms, and the illness is self-limiting.

## CLASSIC TICK-BORNE ILLNESSES

Lyme disease—caused by *Borrelia burgdorferi*. The classic bulls-eye rash (erythema migrans, [Figure 109.2](#)) generally appears within 2 weeks of a tick bite, though as many as 30% of Lyme patients will not have the classic rash. The rash itself begins at the site of the tick bite and becomes larger over several days, usually maintaining distinct borders and associated with little to no pain. Serology is usually negative at the time of the rash. First-line treatment is 14 days of doxycycline.



Rocky Mountain spotted fever (RMSF)—caused by *Rickettsia rickettsii*. The rash of RMFS usually appears within 2 weeks of tick bite. The classic rash of RMFS is petechial and progresses from wrists and ankles inward to the trunk ([Figure 109.3](#)). Unfortunately, this distinctive pattern is absent in up to 70% of patients. The petechial rash appears several days into the course of illness even in those patients with a classical presentation, usually following several days of fever and often a more generalized macular rash. The disease is geographically more diverse than its name suggests, with cases in all but two contiguous states. RMFS is the most lethal tick-borne illness in the United States, and early recognition is essential to reducing morbidity and mortality. Empiric treatment (first line is doxycycline) should be started when RMFS is suspected and not deferred until serologic confirmation.



## KEY POINTS

- Rashes do not always look “classic.”
- Consider rashes within their clinical context—onset, duration, associated factors, vital signs, and overall appearance.
- Although all of the rashes presented here are of viral or bacterial origin, not all rashes are of infectious origin—don’t forget to inquire about medications and exposures.
- Some rashes look significantly different or are difficult to appreciate on darker skin tones.
- Tick-borne illnesses can be acquired in most of the United States and need to be kept on the differential, especially in warmer months.

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## SECTION VI

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# ENDOCRINE/METABOLISM

## A NORMAL BICARBONATE VALUE DOES NOT EXCLUDE AN ACID- BASE DISTURBANCE

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**SETH T. STEARLEY, MD AND IAN BOYD, MD**

Acid-base disturbances are one of the most difficult problems encountered in emergency medicine. It is essential for the emergency provider to quickly discern if a patient has an acid-base disturbance. Misdiagnosis of an acid-base abnormality, or delayed therapy, can lead to serious complications. Plasma bicarbonate concentration ( $\text{HCO}_3^-$ ), pH, and the arterial concentration of carbon dioxide ( $\text{pCO}_2$ ) are critical in acid-base physiology. No value should be evaluated in isolation.

Acid-base disturbances are commonly separated into metabolic or respiratory disturbances. Metabolic disturbances cause a primary change in  $\text{HCO}_3^-$ , whereas respiratory disturbances cause a primary change in  $\text{pCO}_2$ . Although many emergency department (ED) patients have a pure metabolic or respiratory disturbance, it is not uncommon to encounter patients with a mixed acid-base disturbance.

The most common acid-base disturbance encountered in the ED is an anion gap (AG) metabolic acidosis. The anion gap is calculated by the following formula:

$$\text{AG} = \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$$

There are several “unmeasured” cations and anions (i.e., calcium, magnesium and proteins, phosphate, sulfates, lactate) that maintain the electrical neutrality of plasma. An increase in these unmeasured anions

causes  $\text{HCO}_3$  to decrease and results in an AG metabolic acidosis. Often, providers quickly look at the  $\text{HCO}_3$  on a basic metabolic panel as a clue to the presence of an AG metabolic acidosis. Importantly, a significant acid-base disturbance can be present with a normal  $\text{HCO}_3$  level. This is typically seen in the setting of a mixed acid-base disorder. The classic example is a patient who has both vomiting and diarrhea, which causes a concomitant metabolic acidosis and metabolic alkalosis. The diarrhea leads to a loss of bicarbonate and a metabolic acidosis, whereas the vomiting results in a metabolic alkalosis.

Another example is the patient with a mixed metabolic acidosis and respiratory acidosis. In these patients, the respiratory acidosis results in an elevation in  $\text{pCO}_2$ , which then shifts to  $\text{HCO}_3$ . It is important to understand that the kidneys regulate  $\text{HCO}_3$  in the setting of primary respiratory problems. This renal compensation to changes in  $\text{pCO}_2$  can take hours to days to reach equilibrium. Therefore,  $\text{HCO}_3$  can lag in compensation in respiratory problems and therefore be “normal,” when in reality the body is in a state of disequilibrium. It is important to evaluate for appropriate compensation in all acid-base derangements.  $\text{HCO}_3$  concentration, in respiratory disturbances, can be determined with the following equations:

#### **Acute Respiratory Acidosis**

$$\text{Expected } [\text{HCO}_3] = 24 + (\text{pCO}_2 - 40)/10$$

*Usually increased by 1 for every increase of 10 of  $\text{pCO}_2$*

#### **Chronic Respiratory Acidosis**

$$\text{Expected } [\text{HCO}_3] = 24 + 4(\text{pCO}_2 - 40)/10$$

*Usually increased by 4 for every increase of 10 of  $\text{pCO}_2$*

#### **Acute Respiratory Alkalosis**

$$\text{Expected } [\text{HCO}_3] = 24 - 2(40 - \text{pCO}_2)/10$$

*Usually decreased by 2 for every decrease of 10 of  $\text{pCO}_2$*

#### **Chronic Respiratory Alkalosis**

$$\text{Expected } [\text{HCO}_3] = 24 - 5(40 - \text{pCO}_2)/10$$

*Usually decreased by 5 for every decrease of 10 of  $\text{pCO}_2$*

If the expected  $\text{HCO}_3$  does not match the measured  $\text{HCO}_3$ , then there is a concomitant metabolic acidosis or alkalosis present.

Bicarbonate is one of the most important electrolytes in acid-base physiology. Bicarbonate typically fluctuates with acute changes in metabolic

and respiratory disturbances. Importantly, bicarbonate can be normal, or near normal, despite the presence of a significant acid-base disturbance. Failure of recognition can lead to serious complications to our patients and can further complicate care.

## KEY POINTS

- $\text{HCO}_3$ , pH, and  $\text{pCO}_2$  should not be evaluated in isolation.
- Evaluate for appropriate compensation in all acid-base disorders.
- A normal  $\text{HCO}_3$  value should not be used to exclude an acid-base disturbance.
- Mixed acid-base disturbances can often result in a normal  $\text{HCO}_3$ .
- Renal  $\text{HCO}_3$  compensation to respiratory acid-base disorders can take hours to days to reach equilibrium.

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## **DON'T FORGET ABOUT OCTREOTIDE FOR HYPOGLYCEMIA**

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**HALEY M. RAPP, MD AND ERICA B. SHAVER, MD**

Hypoglycemia is defined as a blood glucose concentration  $<50$  mg/dL and accounts for roughly 300,000 emergency department (ED) visits annually in the United States. Most commonly, hypoglycemia occurs in patients with a history of diabetes mellitus. The incidence of hypoglycemia has recently decreased, due to improvements in medications and increased patient education regarding diabetes treatment and control. Notwithstanding, medications account for one of the most common etiologies of hypoglycemia in ED patients.

The sulfonylurea class of medications is a mainstay in the treatment of patients with diabetes. Commonly used sulfonylurea medications include glyburide, glipizide, and glimepiride. These medications increase insulin release by hyperpolarizing adenosine triphosphate-sensitive potassium channels on pancreatic beta cells. This leads to a hyperinsulinemic state, regardless of the blood glucose concentration. This can result in a profound and prolonged hypoglycemia, because the counterregulatory response of the adrenomedullary system to hypoglycemia is often impaired in diabetic patients. The failed counterregulatory response to hypoglycemia is referred to as hypoglycemia-associated autonomic failure. Sulfonylurea medications have varying half-lives that can result in hypoglycemia that is not easily reversed with dextrose alone.

The initial management of any patient presenting with hypoglycemia, regardless of the cause, is rapid supplementation of glucose with either intravenous (IV) dextrose or oral carbohydrate administration. The purpose of IV dextrose administration is to create a relative hyperglycemic state. However, in a patient taking a sulfonylurea medication, this hyperglycemic

state further potentiates pancreatic insulin release, which then perpetuates the vicious cycle of persistent hypoglycemia.

Traditional treatment of sulfonylurea-induced hypoglycemia has been the administration of a continuous infusion of dextrose, sometimes for several days. In recent years, octreotide has been utilized as an adjunctive treatment for sulfonylurea-induced hypoglycemia. Octreotide is a long-acting somatostatin analog that is used in the management of acromegaly, upper gastrointestinal (GI) bleeding due to varices, and metastatic carcinoid symptoms. In the setting of sulfonylurea-induced hypoglycemia, octreotide acts by directly antagonizing the release of insulin from the pancreas. Multiple studies have been performed to assess the safety, efficacy, and role of octreotide in sulfonylurea-induced hypoglycemia. One randomized control trial assessed the efficacy of octreotide plus IV dextrose administration versus dextrose infusion alone and found that patients who received octreotide had higher serum glucose concentrations and fewer hypoglycemic events than did patients who received dextrose alone. Similar results have been found in numerous retrospective analyses. The recommended dose of octreotide for sulfonylurea-induced hypoglycemia is 1 to 2 mcg/kg given via the IV or subcutaneous (SC) route every 8 hours for three doses. Side effects of this medication are generally mild and predominantly limited to GI upset (nausea, vomiting, diarrhea). Although more serious side effects such as hypertension, arrhythmia, and syncope have been reported, these side effects are typically seen in chronic use and are not usually reported in the short-term use of octreotide for sulfonylurea-induced hypoglycemia.

Based on currently available evidence based data, patients who present to the ED with suspected, or known, sulfonylurea-induced hypoglycemia or hypoglycemia refractory to standard IV dextrose administration should be given octreotide.

## KEY POINTS

- Review medications in any ED patient who presents with hypoglycemia.
- Sulfonylurea-induced hypoglycemia can be profound and prolonged.
- Octreotide antagonizes the release of insulin from the pancreas.
- The dose of octreotide in patients with sulfonylurea-induced hypoglycemia is 1 to 2 mcg/kg IV or SC every 8 hours for 24 hours.
- The primary side effects of octreotide are nausea, vomiting, and diarrhea.

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# **PITFALLS IN THE MANAGEMENT OF DKA**

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**ANTHONY ROGGIO, MD**

Diabetic ketoacidosis (DKA) is a critical metabolic derangement that is commonly precipitated by acute illness (i.e., infection, myocardial infarction, stroke) or medication noncompliance. The American Diabetic Association and the International Society for Pediatric and Adolescent Diabetes provide clinical guidelines for the management of patients with DKA. Notwithstanding, these guidelines are complex and can be difficult to follow. Given the mortality associated with DKA, it is imperative for the emergency provider (EP) to be knowledgeable on several pitfalls that can occur in the emergency department management of these critically ill patients.

## **INTRAVENOUS FLUIDS**

Initial management of the DKA patient should begin with intravenous fluid administration. Current guidelines recommend an initial bolus of 15 to 20 mL/kg of 0.9% normal saline. It is important to note that 0.9% normal saline contains supraphysiologic concentrations of chloride. Hyperchloremia is believed to be an inflammatory stimulus and is associated with adverse effects on the renal, pulmonary, cardiovascular, and splanchnic organ systems. Moreover, the strong ion difference of 0.9% normal saline is zero. Thus, 0.9% normal saline will reliably induce a hyperchloremic metabolic acidosis and potentially worsen the already disturbed acid-base balance in patients with DKA.

Recently, balanced solutions (i.e., Plasma-Lyte) have been promoted as better fluid choices in the resuscitation of critically ill patients. Balanced solutions have lower concentrations of chloride and use organic ions as a



substitute for bicarbonate. Studies that have compared the use of balanced solutions to 0.9% normal saline in DKA patients have demonstrated a more rapid closure of the elevated anion gap, increased mean arterial pressure, and more rapid resolution of acidemia. Depending on the specific fluid, balanced solutions also contain varying concentrations of electrolytes (i.e., potassium, calcium, magnesium). While there are no current randomized trials that demonstrate improved mortality with the use of balanced solutions, they are a viable alternative intravenous fluid solution to 0.9% normal saline in the management of DKA.

After an initial fluid bolus, maintenance fluids should be continued at a rate of 250 to 500 mL/hour. A balanced solution should be considered for patients with hyponatremia, whereas 0.45% normal saline should be considered in patients who are hypernatremic or have normal sodium levels.

## INSULIN

The administration of exogenous insulin is essential in the management of DKA. Insulin reverses the ketogenesis that occurs in DKA and corrects the metabolic acidosis. DKA patients can be stratified into mild, moderate, or severe based on the mental status, pH, and serum bicarbonate level. This stratification is listed in [Table 112.1](#).

| TABLE 112.1 DKA AND STRATIFICATION LEVELS |          |              |             |
|---|----------|--------------|-------------|
|   | MILD     | MODERATE     | SEVERE      |
| pH  | 7.25–7.3 | 7–7.24       | <7          |
| Bicarb                                    | 15–18    | 10–14        | <10         |
| Sensorium                                 | Alert    | Alert/drowsy | Stupor/coma |

Historically, insulin has been given as a bolus dose of 0.1 U/kg, followed by an insulin infusion. This administration strategy was felt to achieve adequate serum levels of insulin in a short period of time. Recent literature, however, has demonstrated that an initial bolus dose of insulin is not required, provided that a continuous infusion is started at a rate of at least 0.1 U/kg/h. In addition, recent evidence has suggested that patients with mild or moderate DKA can be safely managed with intermittent subcutaneous doses of short- or medium-acting insulin preparations instead of an insulin infusion. Importantly, subcutaneous insulin administration for DKA requires further study and is not indicated in the treatment of patients with severe

DKA.

Pediatric DKA patients are at risk for cerebral edema. Initiation of insulin within the first hour of treatment or prior to fluid resuscitation has been identified as a risk factor for cerebral edema. For this reason, an insulin bolus should not be administered to pediatric DKA patients. Pediatric patients should receive 1 or 2 boluses of intravenous fluids (20 mL/kg) prior to the administration of any insulin.

Insulin administration should continue until the acidosis of DKA has resolved. Once the blood glucose reaches a value of  $\sim 250$  mg/dL, the type of intravenous fluid should be changed to one that contains dextrose, in order to maintain normoglycemia while the patient is treated with insulin. Once the acidosis has resolved and the anion gap is closed, a long-acting insulin should be administered via the subcutaneous route. The insulin infusion should be continued for  $\sim 1$  to 2 hours after administration of the long-acting insulin in order to prevent rebound hyperglycemia or ketosis once the infusion is stopped.

## POTASSIUM

Many patients with DKA will have elevated levels of potassium, due to electrolyte shifts from the acidosis. However, the majority of patients are actually hypokalemic, due to loss of potassium in the urine from an osmotic diuresis. Importantly, potassium levels will decrease even further with the administration of insulin. Potassium repletion should begin when serum potassium levels are below 5 mEq/L. If serum potassium levels are  $< 3.5$  mEq/L, potassium should be given prior to insulin administration in order to prevent life-threatening hypokalemia.

## SODIUM BICARBONATE

Contrary to conventional wisdom, the administration of sodium bicarbonate may worsen intracellular acidosis and is a risk factor for cerebral edema in pediatric DKA patients. Furthermore, bicarbonate has not been shown to improve patient-centered outcomes in the management of DKA. As a result, sodium bicarbonate is not recommended in the management of DKA, with the possible exception of cardiac arrest secondary to profound acidosis.

### KEY POINTS

- Consider balanced fluids in the resuscitation of patients with DKA.
- An initial bolus dose of insulin is no longer recommended prior to the initiation of an insulin infusion.
- Do not administer a bolus of insulin to pediatric DKA patients.
- Do not administer insulin until the potassium level is  $>3.5$  mEq/L.
- Sodium bicarbonate is not indicated in the treatment of patients with DKA.

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# **DO NOT RELY ON ORTHOSTATIC VITAL SIGNS TO DIAGNOSE VOLUME DEPLETION**

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**ANAND K. SWAMINATHAN GORDON WU , MD, MPH ,  
MD**

Orthostatic blood pressure measurements are commonly thought to be useful in the assessment of intravascular volume status. Orthostatic hypotension (OH) is defined as:

- 1) A reduction in systolic blood pressure (SBP) of at least 20 mm Hg
- 2) A reduction in diastolic blood pressure (DBP) of at least 10 mm Hg
- 3) An increase in heart rate (HR) of at least 30 beats per minute (bpm)

To meet the definition of OH, one of the above criteria must be met when measured at least 3 minutes after standing from a supine position. In addition, patients should also experience symptoms of OH. These symptoms can include light-headedness, dizziness, blurred vision, nausea, palpitations, headache, or weakness that resolves with recumbence.

OH is very common. Data from large studies on unselected elderly nursing home patients demonstrate that the rate of OH ranges from 28% to 50%. Studies in adolescents show similar rates, with ~44% of patients exhibiting orthostatic changes. It is important to remember that OH is simply an exam finding and not a disease.

In an attempt to clarify the significance of OH, Raiha and colleagues performed a prospective cohort study. The authors demonstrated that systolic, or mean, blood pressure changes with standing did not predict

mortality at 10 years. However, a decrease in DBP of at least 10 mm Hg with standing was associated with an increase in vascular mortality (odds ratio of 2.7). This association disappeared in multivariate analysis when the authors adjusted for underlying conditions, such as cardiovascular disease. They hypothesized that patients with more labile DBPs likely had significant comorbid conditions that predisposed them to events like myocardial infarction and stroke.

To further investigate the efficacy of OH as a marker of intravascular volume depletion, Witting and colleagues performed tilt table testing on healthy volunteers after blood donation of <600 mL. In adult patients younger than 65 years of age, a change in HR of >20 bpm or a change in SBP >20 mm Hg had a sensitivity of 47% and a specificity of 84% for volume depletion. Sensitivity and specificity values were similar in patients older than 65 years of age. The results of this study suggest that a finding of OH, either by a drop in SBP or increase in HR, is not specific enough to state with confidence that moderate volume depletion is present. Importantly, just because the patient does not have OH, it does not mean they have a normal intravascular volume. In addition, emergency providers often overestimate the utility of OH in elderly patients. As Witting and colleagues have shown, there is no statistically significant difference between OH in the elderly compared with younger patients as a clinical predictor of volume status.

Building on the work of prior studies, McGee and colleagues assessed the utility of OH symptoms. In their systematic review, the authors demonstrated that the complaint of symptomatic OH had little predictive value in regard to mild to moderate volume loss. However, in patients with severe blood loss (600 to 1,100 mL), there was a dramatic increase in sensitivity (97%) and specificity (98%) in patients who were unable to stand for vital signs measurements secondary to severe dizziness. In this subset of severely symptomatic patients, the inability to stand served as an excellent predictor of severe volume loss. Otherwise, simply the complaint of nausea or dizziness with standing was not clinically useful and should not be routinely used as a measure of intravascular volume status.

## KEY POINTS

- Patients should be standing for at least 3 minutes before orthostatic vital signs are measured.
- OH is very common. Up to 55% of patients may have vital sign changes consistent with OH. This does not mean that they have

intravascular volume depletion.

- OH measurements are neither sensitive nor specific in mild to moderate intravascular volume depletion.
- Orthostatic vital sign measurements in the elderly patient are not a useful clinical predictor of volume status.
- If the patient is symptomatic and unable to stand for vital sign measurements, the patient is more likely to have intravascular volume loss of at least 600 mL.

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# HHS: WHEN HIGH SUGARS HAVE GOT YOU DOWN!

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**STEPHANIE LAREAU, MD, FAWM, FACEP**

Though diabetic ketoacidosis (DKA) is often the first endocrine emergency considered in the patient with hyperglycemia, it is critical for the emergency provider (EP) to include hyperglycemic hyperosmolar state (HHS) in the differential diagnosis. Failure to consider HHS can result in significant delays in treatment and, ultimately, increased patient morbidity and mortality for this critical condition.

The diagnostic criteria for HHS include a serum glucose value  $>600$  mg/dL, serum osmolality  $>320$  osm/kg, a pH above 7.3, a serum bicarbonate  $>15$  mEq/L, and the absence of ketonuria. In contrast to DKA, many patients with HHS will present with symptoms of neurologic dysfunction. These symptoms can include altered mental status, lethargy, seizure, or unilateral deficits that mimic a stroke. Since HHS is more common in elderly patients with Type II diabetes, it can sometimes be difficult to determine if mental status changes are acute, especially in those with dementia. Furthermore, debilitated nursing home patients are at increased risk of HHS. These patients often have impaired access to hydration or are receiving numerous medications that can alter sensorium. It is critical to maintain a high index of suspicion for HHS in hyperglycemic patients with altered mental status, confusion, or lethargy.

The emergency department evaluation of patients with HHS should include a search for the precipitating etiology. The most common precipitant of HHS is infection (i.e., pneumonia, urinary tract infection, sepsis). Other precipitants include myocardial infarction, stroke, medications, renal failure, and head injury (i.e., subdural hematoma). As a result, the emergency department evaluation may include an electrocardiogram, computed

tomography of the head, chest radiograph, urinalysis, and laboratory studies (i.e., complete blood count, comprehensive metabolic panel, troponin, medication levels, lactate).

Patients with HHS have significant intravascular volume depletion. In fact, the average fluid deficit in HHS is ~9 L! Therefore, appropriate fluid resuscitation is critical. Patients who develop HHS often have numerous comorbid conditions, including cardiac and renal dysfunction. As a result, they may not be able to tolerate rapid, large volume resuscitation. Rapid administration of fluids can lead to pulmonary edema and respiratory compromise. An isotonic crystalloid should be administered and the patient monitored closely for signs of fluid overload.

Despite the absence of ketoacidosis and an elevated anion gap, patients with HHS benefit from an insulin infusion to control hyperglycemia. An insulin infusion is initiated at 0.1 U/kg. In contrast to patients with DKA, conversion to a dextrose infusion is usually not required as hyperglycemia is corrected. Patients can be transitioned to subcutaneous insulin treatment when they are able to tolerate oral nutrition. Given the need for close monitoring to prevent hypoglycemia, hypokalemia, and volume overload, patients with HHS are typically admitted to an intensive care unit or intermediate care unit. It is important that the inpatient team continues the evaluation for the precipitating cause of the HHS. Finally, medication adjustments and patient and caretaker education are provided on discharge to prevent recurrence of the condition.

## KEY POINTS

- Consider HHS in the hyperglycemic patient with altered mental status.
- The average fluid deficit in patients with HHS is ~9 L.
- Rapid administration of fluids can lead to pulmonary edema and respiratory compromise.
- An insulin infusion is initiated at 0.1 U/kg for patients with HHS.
- In contrast to patients with DKA, conversion to a dextrose infusion is usually not required as hyperglycemia is corrected.

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# **DO NOT OVER TREAT HYPO- OR HYPERNATREMIA**

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CHANG, MD**

Sodium disturbances are common in the emergency department and can easily stump the emergency provider (EP). Treatment of sodium disorders is based on the lab value, the time course of illness, and the etiology. Mismanagement of the patient with hypo- or hypernatremia can quickly worsen the patient's outcome. The following chapter discusses critical pearls in the treatment of patients with hypo- and hypernatremia.

## **HYPONATREMIA—ADD A PINCH OF SALT**

Hyponatremia is defined as a serum sodium value  $<135$  mEq/L. Hyponatremia can cause cerebral edema, which may lead to cerebral herniation. The clinical presentation of patients with hyponatremia can range from asymptomatic to seizure and coma. All patients should be evaluated for the cause of hyponatremia, as this will assist in management decisions. For example, postoperative and intracranial etiologies for hyponatremia often require urgent correction. It is also important to determine the time course of illness. Patients who have a sudden drop in sodium levels are likely to be more symptomatic and require urgent treatment than those patients who have a slower decrease in sodium levels. Patients with seizure or coma due to hyponatremia require immediate treatment. Asymptomatic patients rarely require emergent therapy.

For patients who require emergent treatment (seizure, coma), administer a 100 mL bolus of hypertonic saline over 10 minutes. The goal is to increase the serum sodium level by 4 to 6 mEq/L over 6 hours. An increase in sodium

by 4 to 6 mEq/L will alleviate most symptoms and prevent cerebral herniation. It is important not to increase sodium levels by more than 8 mEq/L in 24 hours. Overcorrection of sodium can easily occur and cause significant adverse effects. Correction by more than 8 mEq/L in the first 24 hours can increase the risk for osmotic demyelination syndrome, seizure, and cerebral herniation. If patients deteriorate from too rapid of a correction of hyponatremia, desmopressin with D5W should be administered to lower sodium values.

For the patient who does not require emergent treatment, hyponatremia should be treated at a much slower rate. In these patients, it is important to begin with an assessment of intravascular volume status. Patients with hypovolemia or euvolemia may simply need fluid administration. These patients can receive 0.9% normal saline with free water restriction in addition to treatment of the etiology of hyponatremia. For hypervolemic patients, furosemide can be administered along with fluid restriction for treatment.

## **HYPERNATREMIA—WATER IT DOWN**

Hypernatremia is defined as a serum sodium >145 mEq/L. Coma, seizure, and death can result from the shrinkage of cerebral cells as they lose free water. As serum osmolality increases, patients develop excessive thirst, weakness, agitation, ataxia, and neurologic deficits.

Most patients with hypernatremia have chronic hypernatremia that has developed over 48 hours or longer. It is important not to rapidly normalize the serum sodium level. Because of the time required for cerebral adaption to elevated sodium, patients with chronic hypernatremia are at significant risk for cerebral edema with rapid correction. Instead, treatment should aim to lower the serum sodium level by 10 mEq/L over 24 hours. When the serum sodium level is >154 mEq/L, start with 0.9% normal saline rather than a more hypotonic fluid. Once the serum sodium level falls below 154 mEq/L, a hypotonic solution, such as D5W or 0.45% normal saline, can be used. In order to determine the amount of fluid that should be administered, it is important to calculate the free water deficit. The free water deficit can be calculated using the following formula:

$$\text{Free water deficit (FWD)} = 0.6 \times \text{Weight (kg)} \times \left[ \left( \frac{\text{Current Na}}{140} \right) - 1 \right]$$

In patients with chronic hypernatremia, the free water deficit should not be replaced all at once. To select an infusion rate for hypotonic fluid in the patient with chronic hypernatremia, use the following formulas:

$$\text{Water replacement expected in 24 hours} = (\text{FWD} \times 10 \text{ mEq/L}) / (\text{Serum sodium} - 140)$$

$$\text{Hourly infusion rate} = \text{Water replacement expected} / 24 \text{ hours}$$

It is important to search for and treat the etiology of hypernatremia while replacing the free water deficit. Despite appropriate therapy, hypernatremia may not correct appropriately if the patient is having continued loss of free water. Be sure to monitor electrolytes frequently after the initiation of therapy. If serum sodium levels are not improving after 4 hours of therapy, recalculate the free water deficit and adjust the infusion rate. If the patient's sodium has corrected too quickly (at a rate that will decrease the serum sodium by more than 10 mEq in 24 hours), stop the infusion and recheck serum sodium in 2 to 4 hours. One potential reason for the failure of sodium to correct appropriately is the use of D5W. This fluid can cause hyperglycemia and free water loss due to glycosuria. For patients who fail to correct appropriately and have received D5W for free water replacement, change the intravenous fluid to 0.45% normal saline.

## KEY POINTS

- Treatment of sodium disorders depends on the cause, time course of illness, and severity of symptoms.
- For patients who require emergent treatment of hyponatremia, administer a 100-mL bolus of hypertonic saline over 10 minutes.
- When correcting hyponatremia, the goal is to correct by 4 to 6 mEq in the first 24 hours.
- Calculate the free water deficit in patients with hypernatremia. Do not replace the free water deficit all at once, as this can result in cerebral edema.
- Treatment of hypernatremia should aim to lower the serum sodium level by 10 mEq/L over 24 hours.

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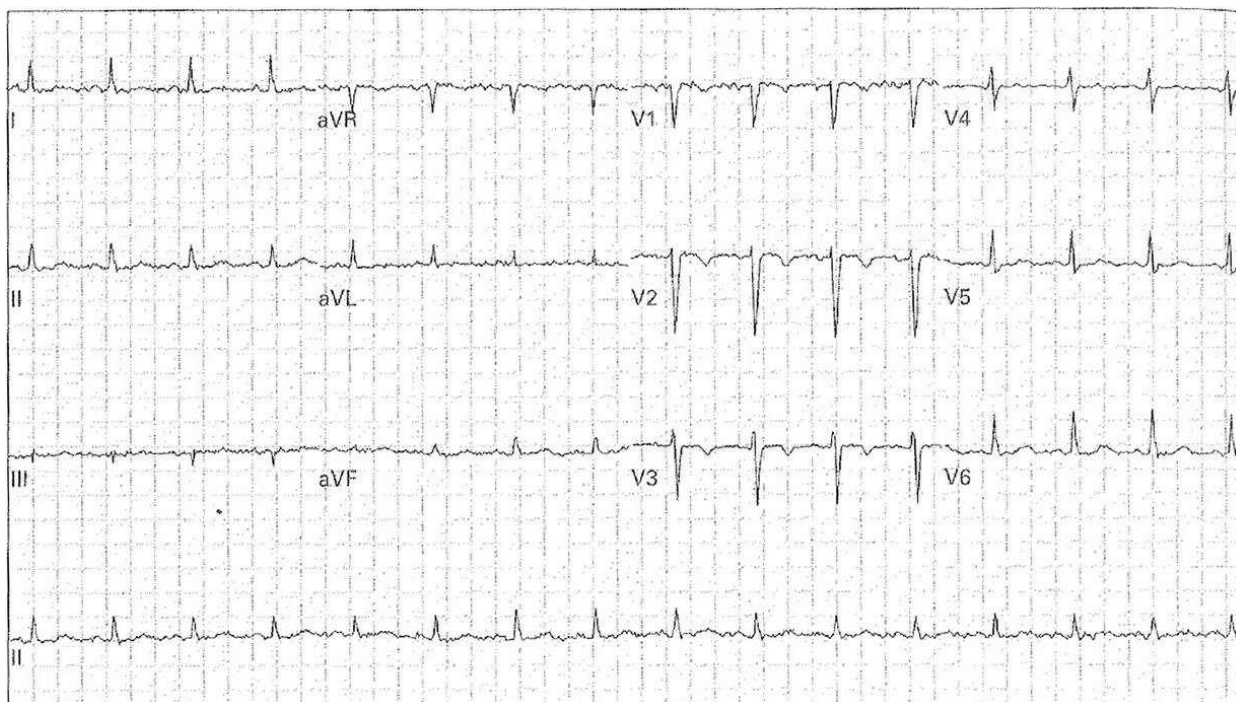
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## A 3-PRONGED APPROACH TO THE TREATMENT OF HYPERKALEMIA

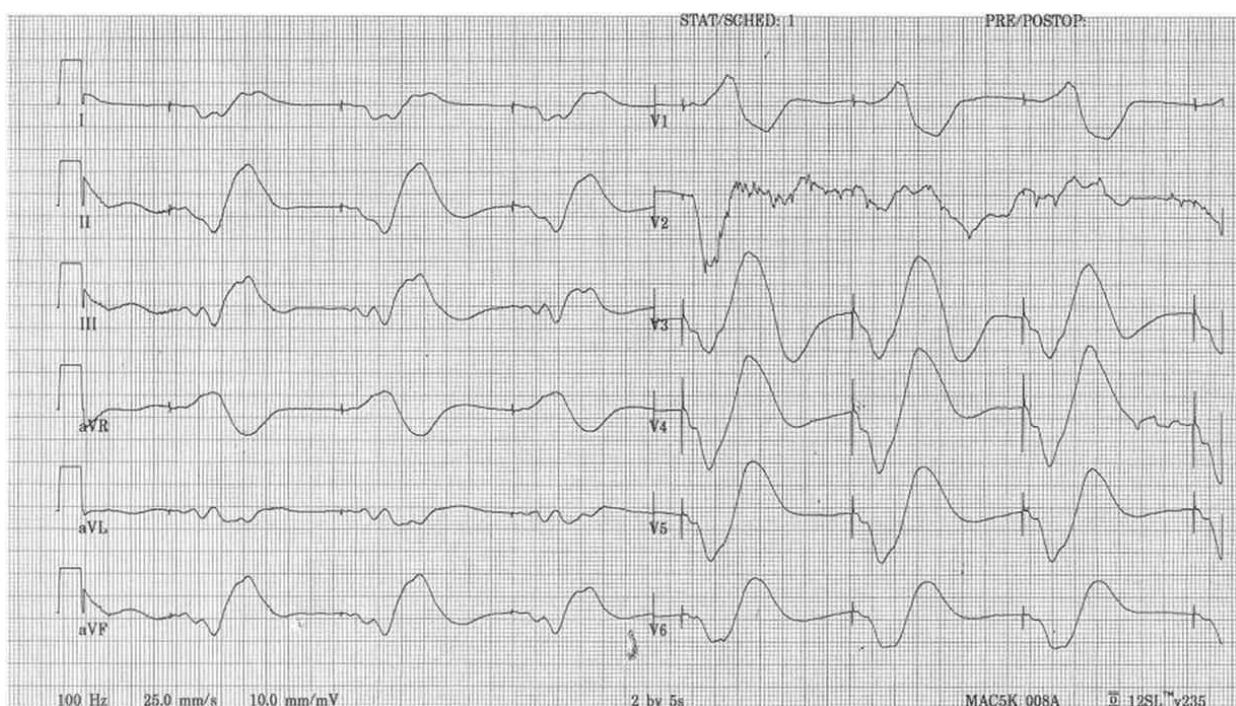
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**ERICA B. SHAVER, MD AND CHRISTOPHER S. KIEFER,  
MD**

Disorders of potassium ( $K^+$ ) regulation are commonly encountered in the emergency setting. Hyperkalemia, defined as a  $K^+$  level  $>5.0$  mEq/L, is the most lethal electrolyte disorder. Given that  $K^+$  is the body's major intracellular cation, even small shifts across cellular membranes can lead to an array of symptoms including nausea, fatigue and muscle weakness. As  $K^+$  levels increase, cardiac membrane instability, electrocardiogram (EKG) changes, and lethal arrhythmias can occur. Hyperkalemia is diagnosed by serum measurement or suggested by classic EKG abnormalities. Classic EKG changes associated with hyperkalemia include peaked T waves ([Figure 116.1](#)), a widened QRS complex, and bradycardia that can lead to the classic "sine wave" morphology ([Figure 116.2](#)).



**Figure 116.1** Peaked T waves in hyperkalemic patient.



**Figure 116.2** “Sine wave” pattern in a profoundly hyperkalemic patient.

Never delay treatment of a patient with classic EKG signs of hyperkalemia while waiting for a serum  $K^+$  level. If concerning EKG changes are noted, it is imperative to initiate treatment to prevent cardiac arrhythmias and circulatory collapse. Similarly, the treatment of a patient

with classic EKG changes should not be delayed due to a serum  $K^+$  level that may not correlate. Patients with normal  $K^+$  levels at baseline generally exhibit EKG changes earlier compared with patients who have chronically elevated potassium levels. These patients often tolerate higher levels before EKG changes are noted. Importantly, EKG abnormalities alone are not indicative of the degree of  $K^+$  elevation.

This chapter will discuss a simple 3-pronged approach to the treatment of hyperkalemia: stabilize, redistribute, and reduce.

## **STABILIZE**

Calcium is the cornerstone of cardiac membrane stabilization in patients with EKG changes associated with hyperkalemia. Importantly, calcium does not change serum  $K^+$  levels. Nonetheless, failure to administer calcium can lead to life-threatening arrhythmias from hyperkalemia. Calcium should be administered prior to the results of serum  $K^+$  levels, especially in patients with chronic kidney disease. Two calcium formulations are commonly given for hyperkalemia: calcium chloride and calcium gluconate. Given the potential for peripheral vein injury, calcium chloride should be administered through a central venous line. Calcium chloride contains three times the elemental calcium as does calcium gluconate. As a result, calcium gluconate requires higher doses for similar clinical effect.

## **REDISTRIBUTE**

Redistribution of serum  $K^+$  is facilitated by administration of albuterol, insulin with glucose, and sodium bicarbonate. Redistribution measures do not eliminate  $K^+$  from the body, but rather shift  $K^+$  to the intracellular compartment.

Albuterol shifts  $K^+$  across cell membranes via a secondary messenger system. Recent studies have reported a decrease of  $K^+$  by  $\sim 0.6$  to  $1.0$  mmol/L in patients given high-dose (10 to 20 mg) nebulized albuterol. Standard doses (5 mg) of nebulized albuterol can be helpful but do not achieve optimal effects.

Insulin shifts extracellular  $K^+$  across the cell membrane via the Na-K ATPase enzyme. In order to avoid hypoglycemia associated with insulin administration, intravenous dextrose is administered concomitantly with 10 units of regular insulin in patients with a serum glucose  $< 250$  mg/dL. It is important to note that insulin is cleared via the kidney. Patients with renal



insufficiency may have delayed clearance of insulin and require additional dextrose to prevent hypoglycemia.

The use of sodium bicarbonate in the treatment of acute hyperkalemia remains controversial. A recent literature has questioned the efficacy of bicarbonate at lowering serum  $K^+$  levels. A recent Cochrane review reported that the evidence for bicarbonate use in hyperkalemia is equivocal. An additional recent review emphasized that there is no significant decrease in serum  $K^+$  with bicarbonate and advised against routine usage.

## REDUCE

Total body serum  $K^+$  can be reduced through hemodialysis, increased urinary excretion, or a binding resin in the stool. Hyperkalemic patients who present with fluid overload and normal renal function may benefit from loop diuretic medications. Loop diuretics work on the loop of Henle and increase  $K^+$  excretion in the urine. Other potassium-depleting diuretics, such as thiazide diuretics, are not as effective as loop diuretic agents.

Hyperkalemic patients with hypovolemia and acute kidney injury and normal urine output may benefit from intravenous fluid resuscitation. Fluid administration in this group of patients may reduce serum  $K^+$  through dilution and potassium excretion through improved renal perfusion.

Sodium polystyrene (Kayexalate) is a cation exchange resin administered orally or rectally that exchanges sodium for  $K^+$  and eliminates  $K^+$  via the gastrointestinal tract. The evidence advocating the use of Kayexalate to treat hyperkalemia originates from a single study published in the 1960s that demonstrated a reduction in serum  $K^+$  in patients with acute and chronic renal failure. Recent literature, however, recommends against routine administration of Kayexalate in the hyperkalemic patient due to an unpredictable reduction of serum  $K^+$  and an increased risk of colonic necrosis.

Oliguric or anuric patients with hyperkalemia will require hemodialysis to definitively remove  $K^+$  from the body. Early consultation with nephrology for hemodialysis should be obtained in patients with oliguria, anuria, or end-stage renal disease.

## KEY POINTS

- Never delay treatment of a patient with classic EKG signs of hyperkalemia while waiting for a serum  $K^+$  level.
- Calcium chloride contains three times the elemental calcium as does calcium gluconate.
- High-dose albuterol (10 to 20 mg) can decrease  $K^+$  by  $\sim 0.6$  to 1.0 mmol/L.
- Recent literature recommends against the routine administration of sodium polystyrene.
- Consult nephrology early for hemodialysis in patients with oliguria, anuria, or end-stage renal disease.

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## KNOW HOW TO RECOGNIZE AND TREAT THYROID STORM

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Thyroid storm is an extreme version of hyperthyroidism with a mortality rate that approaches 30%, even with treatment. The recognition and management of thyroid storm can be difficult in the emergency department (ED). Often, patients present with nonspecific symptoms that can easily be misdiagnosed as more common ED conditions (i.e., sepsis). The following chapter will focus on the clinical presentation, diagnosis, and management of patients with thyroid storm.

Recall that the pituitary gland secretes thyroid-stimulating hormone (TSH), which prompts the thyroid gland to secrete thyroid hormone. Most thyroid hormone is released from the thyroid gland as thyroxine (T4), the physiologically inactive form, while the remainder is released as triiodothyronine (T3), the active form. T4 is converted to T3 in the peripheral tissues. When enough thyroid hormone is present, further release of TSH is inhibited. Under normal circumstances, this feedback mechanism remains in balance. When this balance becomes interrupted, the thyroid gland can become over- or underactive.

Thyroid storm typically presents with elevated temperature, hypertension, tachycardia (often beyond that expected for the elevated temperature), and altered sensorium. The altered sensorium is usually hyperactivity and can vary anywhere from feelings of anxiety to coma. These signs and symptoms can be easily overlooked, especially in the evaluation of a patient with vague complaints. Additional clinical findings include tremors, diaphoresis, thinning hair, exophthalmos, and goiter. Patients with thyroid storm may also manifest signs and symptoms of high-output congestive heart failure, such as pulmonary edema, jugular venous distention, and increased

dyspnea on exertion. Some would call thyroid storm the “great imitator,” as it can present similarly to other more common ED diagnoses.

The diagnosis of thyroid storm should be based on the clinical presentation. Scoring tools for thyroid storm have been published, but no tool has been shown to be superior and these tools are not commonly utilized in the ED. The diagnosis is confirmed with thyroid function testing; TSH, free T3, and free T4 levels should be obtained. Patients with thyroid storm usually have a low TSH level and elevated T3, T4, or both. Though exceedingly rare, a TSH-producing tumor can demonstrate a normal, or elevated, TSH level in conjunction with a high T4 and T3. This tumor should be considered in patients with these lab findings and signs and symptoms of thyrotoxicosis.

The ED management of thyroid storm includes decreasing circulating thyroid hormone levels, reducing the effects of thyroid hormone, treating the presumed etiology, and providing supportive care. These treatment steps should be initiated prior to the results of confirmatory laboratory tests in patients with thyroid storm. Initial management begins with the administration of beta-blocker medications. During thyroid storm, there is an increased expression of beta1-adrenergic receptors, which results in many of the clinical manifestations. Though any beta-blocker can be used, propranolol is generally the medication of choice, as it also blocks the peripheral conversion of T4 to T3. The emergency provider should target a heart rate <100 bpm with the use of a beta-blocker agent. Heart rate control will also improve hemodynamics in patients with high-output heart failure due to thyroid storm. If patients are intolerant to beta-blockers, nondihydropyridine calcium channel blockers may be used with similar efficacy. Importantly, these agents will not block the peripheral conversion of T4 to T3.

Antithyroid medications (i.e., thionamides, iodine) should be administered after a beta-blocker agent has been given. The thionamides, propylthiouracil (PTU) and methimazole, block new thyroid hormone synthesis and should be administered before iodine. Both are administered orally and have been shown to be equally effective. Similar to propranolol, PTU blocks the peripheral conversion of T4 to T3. Iodine will further inhibit thyroid hormone synthesis but should be given at least 1 hour after the administration of a thionamide medication. If iodine is administered before a thionamide agent, it will provide substrate for new hormone synthesis and increase hormone levels. Lastly, glucocorticoid medications block the peripheral conversion of T4 to T3 and should be administered to patients with life-threatening features of thyroid storm.

A critical component in the ED treatment of thyroid storm is temperature management in the setting of fever. A common pitfall is to provide active cooling through the use of cooling blankets, cooled intravenous fluids, and ice packets. However, active cooling methods are contraindicated in thyroid storm. Active cooling will precipitate peripheral vasoconstriction and can worsen hypertension. Temperature management in thyroid storm should consist only of passive cooling techniques, such as a reduction in room temperature and the removal of clothing.

## KEY POINTS

- Consider thyroid storm in the patient with altered mental status and fever.
- Begin treatment for thyroid storm while awaiting confirmatory lab test results.
- Propranolol is the recommended beta-blocker agent of choice and can inhibit the peripheral conversion of T4 to T3.
- Provide iodine therapy at least 60 minutes after the administration of PTU or methimazole.
- Passive cooling is the preferred method for temperature management in patients with thyroid storm.

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# **UNDERSTAND THE ROLE OF MAGNESIUM IN THE TREATMENT OF HYPOKALEMIA**

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Hypokalemia is a common electrolyte abnormality seen in the general population. Causes of hypokalemia include decreased potassium intake, increased entry of potassium into cells, increased gastrointestinal loss, and increased urinary loss. Hypokalemia has many clinical manifestations, some of which can be life threatening and include cardiac arrhythmias and respiratory depression. The primary treatment for hypokalemia is potassium supplementation, along with the identification and correction of the cause of hypokalemia. Unfortunately, exogenous potassium supplementation does not always replete potassium to normal levels. The most common reason for the failure to reach normal potassium levels is that an inadequate amount of potassium is administered for the level of hypokalemia. Another common cause of hypokalemia that is refractory to supplementation is hypomagnesemia. It is essential for the emergency provider to normalize magnesium levels in order to treat hypokalemia.

The majority of potassium is stored within the cells of the body. Oral intake of potassium-rich foods or potassium supplements (i.e., potassium chloride, potassium phosphate, potassium bicarbonate, potassium gluconate) can increase potassium levels. Potassium chloride is the preferred method of oral potassium administration, given its rapid absorption. In addition to potassium supplementation, treatment of hypokalemia also includes the prevention of continued potassium loss (gastrointestinal or urinary sources).

Despite appropriate doses of supplemental potassium, it is common to

have difficulty reaching a normal serum level. Hypomagnesemia is a concurrent electrolyte abnormality seen in up to 60% of patients with hypokalemia. Magnesium is absorbed predominantly in the small intestine via active and passive pathways. Magnesium is then filtered through the renal glomeruli. Magnesium can be lost through gastrointestinal or renal secretion. Magnesium deficiency can worsen hypokalemia and cause it to be refractory to treatment.

The majority of potassium secretion occurs in the distal convoluted tubules and the cortical collecting duct, while the majority of potassium reabsorption occurs in the proximal tubule and loop of Henle. Magnesium plays a critical role in potassium homeostasis by decreasing potassium secretion and increasing its excretion within the kidney. In the distal convoluted tubule and cortical collecting duct, potassium is absorbed into the cell across the basolateral membrane via a  $\text{Na}^+/\text{K}^+$ -ATPase pump. Once within the cell, potassium is secreted into the urine via the ROMK channel. Magnesium inhibits this channel and lowers the amount of potassium secreted into the luminal fluid. When the body has a magnesium deficiency, there is no longer inhibition of this channel and potassium is readily secreted into the lumen and excreted in the urine.

Common causes of hypomagnesemia include malnutrition, vomiting, diarrhea, and malabsorption. The most important step is to consider concomitant hypomagnesemia in the setting of hypokalemia that is refractory to treatment. The treatment for hypomagnesemia is magnesium supplementation and identification of the etiology. Once magnesium is repleted, potassium repletion can occur and has a higher chance of success. Normal serum magnesium level ranges from 1.5 to 2.4 mg/dL. In mild to moderate hypomagnesemia (1.0 to 1.5 mg/dL), 1 to 4 g of magnesium sulfate can be administered intravenously (IV) at a maximum rate of 1 g/hour. In severe symptomatic hypomagnesemia, 1 to 4 g of magnesium sulfate can be administered IV over 5 to 60 minutes. Intravenous doses should be decreased by 50% in patients with renal insufficiency. Faster rates of infusion can result in an increase in urine magnesium excretion. Oral preparations of magnesium include magnesium chloride and magnesium oxide. These have limited bioavailability but are useful for outpatient therapy.

## KEY POINTS

- Hypomagnesemia is present in up to 60% of patients with hypokalemia.

- Hypokalemia is difficult to correct before magnesium is replaced.
- Search for the cause of hypokalemia and hypomagnesemia.
- IV replacement is the preferred route to replace magnesium.
- In severe hypomagnesemia, 1 to 4 g of magnesium sulfate can be administered IV over 5 to 60 minutes.

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## KNOW HOW TO INTERPRET THE VENOUS BLOOD GAS

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**JOSHUA (JOSH) NICHOLS, MD AND COREY HEITZ, MD**

The decision to obtain a venous blood gas (VBG) or an arterial blood gas (ABG) can be a challenge for the emergency provider (EP). It is sometimes difficult to know when either test is indicated. Importantly, a VBG can save time, spare the patient the pain of an arterial blood draw, avoid arterial injury, and avoid potential arterial thrombosis and ischemia. Notwithstanding, it is critical for the EP to know how to interpret the results of the VBG. This chapter discusses the correlation of the pH, arterial partial pressure of carbon dioxide ( $\text{PaCO}_2$ ), arterial partial pressure of oxygen ( $\text{PaO}_2$ ), the bicarbonate ( $\text{HCO}_3$ ), and lactate levels obtained with a VBG compared to that of an ABG.

### pH

The pH of the VBG correlates well with the pH of an ABG. In two large meta-analyses, authors demonstrated that the mean difference between the venous and arterial pH is between 0.033 and 0.035. The VBG pH has shown good correlation in patients with acidosis (i.e., diabetic ketoacidosis) and alkalosis, where the pH ranges from 7.05 to 7.61.

There are two clinical instances where the provider must be wary of the pH from a VBG. There are insufficient data to support using venous pH in patients with mixed acid-base disorders. Additionally, few studies have been conducted that correlate venous and arterial pH in the hypotensive patient, who has a systolic blood pressure  $<90$  mm Hg. In one small study, authors showed that the arterial to venous pH difference increased slightly in hypotensive patients compared to normotensive patients; however, this

increase was not statistically significant. Due to the relative paucity of studies that have examined pH from a VBG in the setting of hypotension, the EP should consider an ABG pH in patients with shock.

## **CARBON DIOXIDE**

The partial pressure of carbon dioxide ( $\text{PCO}_2$ ) of the VBG is an important measurement. Unfortunately, the  $\text{PCO}_2$  from a VBG does not correlate well enough with the  $\text{PaCO}_2$  of the ABG to be used simply as a surrogate marker. At normal  $\text{PCO}_2$  levels, the VBG and ABG  $\text{PCO}_2$  do correlate well. This association does not hold well in patients with hypercapnia. However, the  $\text{PCO}_2$  from a VBG can be used to screen for hypercapnia. A venous  $\text{PCO}_2$  value that is  $<45$  mm Hg has been shown to have a negative predictive value of 100% for a  $\text{PaCO}_2$  that is  $>50$  mm Hg. If the VBG  $\text{PCO}_2$  is normal, hypercapnia can reliably be excluded. If the VBG  $\text{PCO}_2$  is  $>45$  mm Hg, the EP should obtain an ABG to measure  $\text{PaCO}_2$  and determine if there is clinically relevant hypercapnia.

## **OXYGEN**

The partial pressure of oxygen ( $\text{PO}_2$ ) from a VBG correlates poorly with the  $\text{PaO}_2$  from an ABG. In one study, authors estimated the VBG  $\text{PO}_2$  was  $\sim 37$  mm Hg less than the ABG  $\text{PaO}_2$ . In this study, the 95% confidence interval was sufficiently wide to make any correlation between the two values. A notable exception to this is in cases of cyanide toxicity, where the EP may see arterialization of the VBG  $\text{PO}_2$  due to binding of cyanide to cytochrome c oxidase. This halts the electron transport chain and prevents conversion of oxygen to water. In this instance, VBG  $\text{PO}_2$  may be within 10% difference of the ABG  $\text{PaO}_2$ .

## **BICARBONATE**

As with pH, the VBG  $\text{HCO}_3$  correlates well with arterial  $\text{HCO}_3$ . In two large meta-analyses, the mean difference between venous and arterial  $\text{HCO}_3$  was 1.03 to 1.41. The small mean difference and a narrow confidence interval make venous estimation of arterial  $\text{HCO}_3$  clinically useful in most cases. However, the EP must be cognizant of the patient's underlying medical conditions. In one study that compared venous and arterial pH in patients

with an exacerbation of chronic obstructive pulmonary disease (COPD) who had hypercapnic respiratory failure, individual arterial and venous  $\text{HCO}_3$  measurements differed by as much as  $-6.24$  to  $+10.0$  mmol/L. This suggests that the  $\text{HCO}_3$  from a VBG may be less useful in these patients. Although not entirely clear, this poor level of agreement may be due to COPD, which causes a baseline chronic metabolic alkalosis combined with an acute respiratory acidosis. This finding highlights the poor understanding of VBG and ABG correlation in mixed acid-base disorders.

## LACTATE

VBG lactate correlates well with arterial lactate at normal levels ( $<2$  mmol/L). A systematic review showed that at normal levels, the mean difference between venous and arterial lactate is 0.25 mmol/L. However, the authors noted that in studies that included hemodynamically unstable trauma patients and patients with higher lactate levels, there was a weaker correlation between arterial and venous lactate.

### KEY POINTS

- The VBG is appropriate for estimating arterial pH and  $\text{HCO}_3$ , unless the patient is hypotensive or there is suspicion of a mixed acid-base disorder.
- The VBG  $\text{PCO}_2$  can be used to screen for hypercapnia. There is poor correlation with  $\text{PaCO}_2$  at values  $>45$  mm Hg.
- The VBG  $\text{PO}_2$  is not clinically useful, except in cases of suspected cyanide toxicity.
- The VBG lactate correlates best with ABG lactate at values  $<2$  mmol/L.
- ABG is preferable to VBG in patients with shock, severe trauma, and mixed acid-base disorders.

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# **KNOW THE INDICATIONS FOR BICARBONATE THERAPY**

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**KIMBERLY BOSWELL, MD**

The use of sodium bicarbonate in the emergency department (ED) has varied over generations. Bicarbonate has been used to treat numerous conditions, such as diabetic ketoacidosis (DKA) and in the prevention of contrast-induced nephropathy (CIN) for patients undergoing computed tomography studies. Recent literature, however, has challenged the utility and safety of bicarbonate administration for several disease processes. Currently, there are only a few indications for bicarbonate use in the ED. This chapter discusses the utilization and controversies of bicarbonate therapy in the ED.

The foundation of DKA treatment includes aggressive fluid resuscitation, electrolyte management, and insulin. Bicarbonate is no longer recommended to treat the acidosis associated with DKA, unless the patient's pH falls below 6.9. If bicarbonate is given for a pH < 6.9, it should be given in small aliquots (100 mEq) and infused over 1 to 2 hours. The venous pH should be checked every 2 hours and bicarbonate should be stopped when the pH is above 7.0. The controversy surrounding bicarbonate therapy in DKA is primarily twofold. First, there is little evidence to support the benefit of its administration, and there are several possible side effects, including a paradoxical decrease in the cerebral pH and a decrease in serum potassium levels. Second, there is evidence to suggest that it decreases the clearance of ketones. Importantly, its use in pediatrics is discouraged due to its association with cerebral edema. The routine use of bicarbonate therapy in DKA should no longer be considered a pillar of treatment and should be reserved for the most critically ill patients.

Lactic acidosis can be the result of numerous disease processes or injuries and reflects hypoperfusion of tissues. The use of sodium bicarbonate

to treat lactic acidosis remains controversial and generally should be considered when the pH is  $<7.1$  and the serum bicarbonate is  $<6$ . Profound acidosis negatively impacts patient hemodynamics through a reduction in cardiac contractility and a decreased response to catecholamines. This, in turn, leads to arteriolar vasodilation and can result in various arrhythmias. For patients with a pH  $> 7.1$ , it is recommended that treatment focus on the etiology of lactic acidosis rather than administration of bicarbonate. Several studies have demonstrated that there is little difference in the administration of bicarbonate compared to saline with respect to cardiac output and mean arterial pressure in the patient with a pH  $> 7.1$ . In fact, it is important to remember that the administration of exogenous bicarbonate can effect electrolyte balance and result in hypocalcemia, hypernatremia, and hypokalemia. In addition, exogenous bicarbonate stimulates arterial and tissue production of carbon dioxide. The goal of bicarbonate treatment in severe lactic acidosis is simply to achieve a pH  $> 7.1$  while simultaneously treating the etiology.

Another controversial topic is the administration of bicarbonate to prevent CIN. The belief that alkalinization protects the kidneys from free radical damage fostered the belief that bicarbonate therapy would be beneficial in CIN prophylaxis. However, the mechanism by which contrast affects the kidney is not well understood. Many randomized trials and meta-analyses have demonstrated equivocal outcomes when bicarbonate was compared with normal saline. In 2012, the Kidney Disease: Improving Global Outcomes Guidelines recommended simple administration of isotonic fluids for volume expansion for CIN prophylaxis. This recommendation remains current.

Severe, life-threatening hyperkalemia remains an important indication for the use of bicarbonate in the ED. Bicarbonate shifts extracellular potassium to the intracellular compartment in order to maintain an electrically neutral environment. Interestingly, there have been no studies that actually demonstrate an immediate or significant benefit (acute change in the serum potassium level) with bicarbonate. It is important to note that administration of bicarbonate should not be the only intervention employed in the acute treatment of hyperkalemia. The administration of calcium, insulin and glucose, and beta-2 agonists is still indicated.

## KEY POINTS

- Bicarbonate should not be used in the routine treatment of DKA,

unless pH drops below 6.9.

- Administer bicarbonate to patients with a severe lactic acidosis (pH < 7.1).
- Bicarbonate therapy can induce hypokalemia, hypernatremia, and hypocalcemia.
- Bicarbonate therapy is not indicated for prophylaxis of CIN.
- Administer bicarbonate to the patient with severe hyperkalemia who is not responding to traditional therapies, such as insulin and glucose and beta-2 agonists.

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# **SECTION VII**

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# **ENVIRONMENT**



## NOT SO FAST! REWARMING THE COLD PATIENT

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**KUBWIMANA MOSES MHAYAMAGURU, MD, EMT-P AND  
CHRISTOPHER G. WILLIAMS, MD, FAWM**

Whoever said “take your time,” “slow is smooth, smooth is fast,” “measure twice and cut once,” and “slowly but surely” may well have been speaking of hypothermia treatment. We will discuss how this sage advice pertains to the hypothermic patient later. First, though, let us review the mechanics of normal thermoregulation and heat transfer from the body.

### **THERMOREGULATION**

A normal body temperature is said to be 36.4°C to 37.5°C. Achieving a normal body temperature is the result of balancing heat production and heat loss. The vast majority of heat leaves the body through the skin via radiation, evaporation, conduction, and convection. The majority of remaining heat loss occurs via respiration.

Thermal regulation is controlled by the anterior hypothalamus. There are factors/conditions that can alter thermoregulation, and they include extremes of ages, endocrine disorders, malnutrition, and hypoglycemia, which all tend to limit heat production. Other things that can interfere with thermoregulation include breakdowns in skin integrity (e.g., burns, road rash) or inappropriate vasodilation in the periphery (e.g., mediations, spinal cord injuries, sepsis). Hypothermia has been defined as a core body temperature  $\leq 35^{\circ}\text{C}$  ( $95^{\circ}\text{F}$ ). It is commonly subdivided into three levels of severity: mild hypothermia ( $32^{\circ}\text{C}$  to  $35^{\circ}\text{C}$  [ $90^{\circ}\text{F}$  to  $95^{\circ}$ ]), moderate hypothermia ( $28^{\circ}\text{C}$  to  $32^{\circ}\text{C}$  [ $82^{\circ}\text{F}$  to  $90^{\circ}\text{F}$ ]), and severe hypothermia, which equals a core temperature  $<28^{\circ}\text{C}$  ( $82^{\circ}\text{F}$ ). At temperatures approaching  $30^{\circ}\text{C}$ , humans

become altered—even to the point of coma—and cardiac dysrhythmias may occur. At 23°C, apnea is common.

## TREATMENT OF HYPOTHERMIA

Noninvasive passive external rewarming tends to be enough for patients with mild hypothermia. A basic thing like covering up the patient with dry clothing in a warm room generally suffices.

For moderate hypothermia, more aggressive external rewarming is indicated with the use of heated blankets, hot pads, hot water bottles, and chemical warmers. In the case of severe hypothermia, invasive rewarming attempts should be undertaken. This includes cardiopulmonary bypass or intravenous rewarming. Intracavitary lavage has grown out of favor.

Patients with mild hypothermia can have intense shivering and cold white/pale skin. Patients with moderate hypothermia have altered mental status in the form of dysphasia, amnesia, confusion, or apathy—symptoms mimicking many pathologies. In addition, they tend to be hyporeflexic and have ataxia or loss of fine motor skills. Patients with severe hypothermia lose complete ability to shiver; they may be delirious or comatose and have fixed and dilated pupils, oliguria, bradycardia, hypotension, and pulmonary edema. Keeping these signs and symptoms in mind is vital. It is not unheard of for a patient to be found with ataxia and decreased level of consciousness, delivered to the ED not shivering, and ending up being intubated for suspicion of other causes of altered mental status (AMS) without considering their core temperature as the possible etiology.

Perhaps the greatest threat in the resuscitation of hypothermic patients is dysrhythmia. This can result in a refractory ventricular fibrillation and is associated with jostling or manhandling the patient. Care should be taken both prehospital and in the resuscitation bay when transferring the patient, placing pads, performing procedures, etc. A second, more insidious threat is the afterdrop phenomenon. This occurs as rewarming of the body causes stagnant, frigid blood in the periphery to circulate back toward the core, leading to a paradoxical drop in core temperature. This author has met Greenlandic Inuits who report witnessing this event frequently. One of these Inuits (who is himself a physician) teaches that arms and lower legs should be kept out of warming blankets and away from heating pads until the core is sufficiently warmed.

It is also important to note that a patient may not necessarily be dead when found down, cold, cyanotic, and without apparent cardiac or respiratory activity. As the saying goes, “a patient is not dead until they’re

warm and dead.” The American Heart Association (AHA) recommends rewarming patients up to 35°C before declaring resuscitative efforts futile and withdrawing support. (Obviously, this only applies to cases where cause of death is ambiguous or potentially environmental). The AHA has a modified AHA algorithm for hypothermic resuscitation. Some things to remember are as follows: administration of code drugs and defibrillation should be withheld until rewarming to at least 28°C is achieved, and acquiring an EKG may be difficult on cold skin, for which the use of pin electrodes is an option.

In addition to VF and afterdrop, some potential complications to keep in mind while rewarming the severely hypothermic patient include hypokalemia and hypophosphatemia, hypoglycemia, rewarming-related hypertension, bladder atony, paralytic ileus, coagulopathy, and rhabdomyolysis. Every patient should be monitored during and after rewarming, with attention paid to the above complications.

## KEY POINTS

- Slow is smooth; smooth is fast: Avoid rough movements and handle the victim gently for all procedures to avoid VF.
- Preexisting or concurrent conditions may be the exacerbating force leading to hypothermia.
- When in doubt, adequately warm the core before the extremities to prevent afterdrop.
- Resuscitation should be continued until the absence of cardiac activity is documented after raising the body temperature to a level of 28°C to 30°C.

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# ACCLIMATIZE OR DIE OR DESCEND

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CLINTON G. KEILMAN, MD

## ACUTE MOUNTAIN SICKNESS

Acute mountain sickness (AMS) is an intolerance to hypoxia that usually occurs in the first few days of travel to altitudes above 8,200 ft. The symptoms can start as early as 2 hours, but rarely after 36 hours at altitude. Rapid rate of ascent, higher altitudes, unacclimatization, increased physical exertion, and genetic predisposition all increase the risk and severity of AMS. The hallmark sign of AMS is headache, which is usually bitemporal, throbbing, and worse with the Valsalva maneuver. Symptoms that can accompany the headache are nausea, vomiting, anorexia, GI disturbance, dizziness, dyspnea on exertion, malaise, and lassitude. AMS typically resolves in 1 to 2 days and is self-limiting and not life threatening. AMS does not have neurologic findings aside from headache. If neurologic findings are present, it is likely that AMS has progressed to high-altitude cerebral edema (HACE). The Lake Louise consensus definition for AMS is the presence of headache and at least one of the following: gastrointestinal problems, fatigue or weakness, dizziness or light-headedness, and difficulty sleeping.

### *Prevention*

As with nearly all medical conditions, prevention is far superior to treatment. The Wilderness Medical Society has presented recommendations for the prevention/prophylaxis of AMS. These include taking  $\geq 2$  days to arrive at 3,000 m and limiting subsequent sleeping elevation to  $< 500$  m/d, acetazolamide 125 mg bid, and/or dexamethasone 4 mg bid. These recommendations are nuanced depending on an individual patient's risk category for high-altitude illness, so the reader is referred to the WMS consensus guidelines for details. The bottom line is that if a patient follows a

proper ascent profile, the use of acetazolamide or dexamethasone can be avoided in most cases.

## ***Treatment***

For mild AMS, stop the ascent to allow time to acclimatize and treat with analgesics and antiemetics for symptomatic relief. Acetazolamide 125 to 250 mg bid can be used to speed up acclimatization or one should descend by 1,600 ft or more until symptoms resolve. To treat moderate to severe AMS, use dexamethasone 4 mg PO, IM, IV q6h, oxygen 2 L/minute, and acetazolamide 250 mg bid. (Think “DOA,” if you don’t want your patients “dead on arrival.”) Descend if possible, but if unable to descend, treat with portable hyperbarics.

## **HIGH-ALTITUDE CEREBRAL EDEMA**

HACE is an encephalopathy with the cardinal symptoms of ataxia and change in consciousness (confusion, somnolence, coma) with focal neurologic signs and seizures being uncommon. AMS progression to HACE usually requires 1 to 3 days, but can happen as quickly as 12 hours. The use of labs can be helpful in ruling out other conditions; however, imaging has a minor role. “Computed tomography (CT) may show compression of sulci and flattening of gyri, and attenuation of signal more in the white matter than gray matter. MRI is more revealing, with a characteristic high T2 signal in the white matter, especially the splenium of the corpus callosum, and most evident on diffusion-weighted images.” The Lake Louise consensus definition for HACE is the presence of one of the following: presence of a change in mental status and/or ataxia in a person with AMS or the presence of both mental status changes and ataxia in a person without AMS.

## ***Treatment***

Treatment for HACE is similar to that for AMS but is more urgent. Immediately descend, and if you cannot descend, treat with portable hyperbarics. A portable hyperbaric bag compressed to two psi is the equivalent of descending 5,250 ft. In addition to descending, treat early with dexamethasone 8 mg PO, IM, or IV and then 4 mg q6h and oxygen 2 to 6 L/min.

## **HIGH-ALTITUDE PULMONARY EDEMA**

High-altitude pulmonary edema (HAPE) usually occurs on day 2 to 4 on

altitudes >8,200 ft and is the most common cause of death at altitude. The Lake Louise consensus defines HAPE as a minimum of two of the following symptoms: dyspnea at rest, cough, weakness or decreased exercise performance, chest tightness, or congestion. In addition, there must be a minimum of two of the following signs: crackles or wheezing in at least one lung field, central cyanosis, tachypnea, or tachycardia. If recognized early, this condition is easily reversible.

## ***Treatment***

The treatment of HAPE is similar to HACE in that you must descend but you must also minimize exertion. If one cannot descend, treat with portable hyperbarics, oxygen 2 to 6 L/min, and add nifedipine 30 mg PO q12h (sildenafil or tadalafil as an alternative) to reduce pulmonary artery pressure and resistance.

## **KEY POINTS**

- Most EM providers will not see this in the ED unless it is a severe case because most will resolve by the time they reach an ED.
- When faced with questions about a patient with symptoms at altitude, descent is always the right answer.
- Early recognition is key to treat and prevent further deterioration of the patient.

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# **AGGRESSIVE COOLING IS (ALMOST) ALWAYS THE CORRECT APPROACH TO THE CRITICAL, ENVIRONMENTALLY HYPERTHERMIC PATIENT**

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**CHRISTOPHER G. WILLIAMS, MD, FAWM**

During a December storm in 1790, local physician, James Currie, stood helplessly from the shore near Liverpool Harbor while a number of American crewmen floundered and succumbed to the 40-degree waters. Unable to maintain their grasp to the surrounding rigging and flotsam, many drowned. Following this ordeal, Dr. Currie began to undertake a series of human experiments involving cold water immersion (CWI). It is from these limited experiments of only a handful of euthermic, healthy subjects that the phenomenon known as the “Currie response” was derived. This argues that an individual placed rapidly into a cold environment will vasoconstrict, shiver, and temporarily maintain or elevate his or her temperature (by 0.1°C to 0.2°C total). These findings crept into modern medical teaching along with the dogma that hyperthermia should only be treated with a few strategically placed ice packs, evaporative cooling with tepid-to-warm water, avoidance of CWI, etc. For about 200 years, the medical community, including emergency physicians, has practiced beneath this well-meant but poorly derived conclusion that cooling measures should be implemented cautiously.

Just as the severity of thermal burns is a function of heat intensity and duration of contact, hyperthermia resulting in end-organ damage should be



viewed similarly. Consequently, aggressive treatments are indicated to limit the amount of time a patient remains hyperthermic. Unlike the treatment of severe hypothermia, when premature and overly aggressive correction can result in afterdrop phenomena, arrhythmias or refreezing injuries, hyperthermic patients tolerate—in fact require—as timely a correction as safely feasible. Time can mean neurons, nephrons, myocytes, and hepatocytes, so cool them, and cool them quickly.

Heat-related illness is the leading cause of morbidity and mortality among US high school athletes, as well as military recruits during training. Heat waves annually kill many thousands, predominantly among the extremes of age and those of lower socioeconomic means. Heat illness in general should be viewed as a spectrum involving minor symptoms such as heat edema, heat rash, heat cramps, heat syncope, or moderate-to-severe symptoms, namely, heat exhaustion, heat injury (implying end-organ damage), and heat stroke (CNS impairment).

Upon arrival to the emergency department, a patient suspected of moderate-to-severe heat illness may require more than simple passive cooling measures. Heat is dissipated via conduction, convection, radiation, and evaporation. Remove all clothing, especially constrictive clothing such as uniforms or football pads. Skin temperatures can be misleading, so core temperature should be obtained either rectally or via a temp-sensing Foley catheter. (Be aware that the rectum is well insulated, so as cooling efforts take effect, the measured temperature may actually lag behind the core temperature.) Typically, a core temperature  $>40^{\circ}\text{C}$  correlates with severe heat injury or heat stroke, but this is not absolute. Hyperthermia + CNS dysfunction = heat stroke. A temperature  $<40^{\circ}\text{C}$  or the presence of sweating should not dissuade a thoughtful clinician from making the diagnosis of heat stroke.

Dehydration is associated with decreased sweat rates and increased core temperatures, so begin efforts to rehydrate. Oral rehydration is sufficient for mild-to-moderate cases where end-organ derangement is not in question. IV fluids, typically crystalloids, are very commonly given in these patients, but should not be given dogmatically; the goal should be euvolemia. Keep in mind that symptomatic exercise-associated hyponatremia may present with similar symptoms as heat exhaustion or heat stroke, such as weakness, ataxia, and altered mentation. Encouraged consumption of free water in hyponatremic patients thought to be merely dehydrated has yielded catastrophic consequences.

So what about this “Currie response”? Studies and practical experience show that in dangerously hyperthermic patients, CWI does not result in

shivering or temperature gains. Whatever peripheral vasoconstriction occurs is insufficient to counteract the effects of aggressive cooling measures. Studies of CWI show cooling rates of hyperthermic patients at 0.2°C/minute to 0.35°C/minute, orders of magnitude faster than other conventional cooling methods. However, unless your patient is an otherwise healthy victim of exertional heat stroke (EHS), the need for monitoring, managing airway, evaluating for comorbid conditions, etc. may make immersive therapy unfeasible.

There are expensive, name-brand options for rapidly cooling the critical patient. These usually come in the form of pads or blankets and have the added benefit of tighter monitoring of core temperature, avoidance of overcooling, and maintaining a desired temperature. (The latter two are not usually a factor in the treatment of environmentally hyperthermic patients.) In many emergency departments, infrequent use and nursing unfamiliarity makes the placement and use of these proprietary systems slow and impractical. Perhaps the most common method of active cooling that does not limit monitoring is the use of evaporation/convection. After clothing is removed, hospital sheets or towels may be loosely draped over the patient and doused with cold water. A box fan or a floor fan (snail fan) should be obtained—call hospital maintenance if necessary—and directed at the patient. As long as active cooling efforts are under way, the sheets should be turned or replaced when warmed up and rewetted when dry. This has been shown to decrease core temperature on average 0.04°C/minute to 0.1°C/minute. It is likely that adding ice packs to the patient augments this cooling rate.

## KEY POINTS

- Hyperthermic injury is a time-sensitive process; so cool the patient!
- Aggressive cooling efforts will not cause critically hyperthermic patients to paradoxically shiver and raise their temperature.
- Evaluate for concomitant dehydration and do not miss hyponatremia.
- Cease active cooling measures when core temperature <39°C.

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## SMOKE INHALATION: COMMONLY OVERTREATED AND UNDERTREATED ASPECTS

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**DENNIS ALLIN, MD, FACEP, FAAEM, FAEMS**

Smoke inhalation is the most common cause of death from fires, increasing the mortality of a 30% total body surface area (TBSA) burn by 70%. Smoke inhalation generally occurs in enclosed spaces, and treatment involves the management of multiple mechanisms of injury including thermal burns from fire and inhalation of superheated gases, direct effects of inhaled chemical irritants, and inhaled substances producing systemic toxicity.

### **THERMAL BURNS**

With few exceptions, the thermal burns in the airway will occur in the oropharynx with the dissipation of heat protecting the lower airways. The signs suggesting upper airway thermal injury include

- 1) Stridor
- 2) Hoarseness
- 3) Carbonaceous sputum
- 4) Visible burns and blistering of the mucosa or face

In patients involved in an enclosed space fire with signs of potential airway involvement, elective intubation should be considered prior to signs of airway obstruction as deterioration of the airway can occur very rapidly and, once present, will make endotracheal intubation nearly impossible due to swelling and constriction of the airway. Methods to evaluate the glottis

include rapid sequence induction, awake laryngoscopy with local anesthesia, and fiberoptic laryngoscopy. Patients with obvious signs of airway thermal injury will likely have concomitant pulmonary injury that will also require early aggressive securing of the airway to manage the acute pulmonary injury as well as to provide necessary pulmonary toilet.

## SYSTEMIC TOXINS

Carbon monoxide is the product of incomplete combustion of carbon compounds and thus a common component of smoke inhalation. Toxicity generally relates to hypoxic stress from binding to hemoglobin and leftward shift of the oxygen dissociation curve. There are, however, additional mechanisms of direct interruption of cellular metabolism as well as immunologic and inflammatory pathologic processes likely unrelated to hypoxia that can evolve over time. These processes affect mainly neuro tissue leading to delayed neurologic sequelae including headaches, motor weakness, balance issues, and cognitive deficits. The diagnosis of carbon monoxide toxicity is suspected in patients with potential exposure to carbon monoxide with headache, dizziness, vomiting, altered mental status, loss of consciousness, severe acidosis, and cardiovascular dysfunction, confirmed by measurement of either venous or arterial carboxyhemoglobin levels. Remember that measurements in the ED may be low and have poor correlation with the level of toxicity due to administration of oxygen by EMS providers. Any HbCO level >10% in a smoker, or >4% in a nonsmoker, is indicative of CO exposure. Once confirmed, the primary treatment is 100% oxygen.

The role of hyperbaric oxygen therapy in carbon monoxide poisoning remains somewhat controversial, but the generally accepted indications to refer a patient for hyperbaric oxygen treatment include prolonged loss of consciousness, neurologic dysfunction, or cardiovascular dysfunction, but it must be understood that the principle purpose of hyperbaric oxygen therapy is the prevention of neurologic sequelae and that the patient should be stable from an airway and pulmonary status. Even then, these patients require a hyperbaric center capable of critical care, and the number of these centers in the United States is decreasing. The Divers Alert Network reports that only 30% of the hyperbaric departments in the United States can take patients 24 hours a day. For these reasons, the treating physician should consider carefully the risk versus benefit of overemphasizing the transfer of a patient for hyperbaric oxygen therapy prior to managing the life-threatening complications of thermal burns to the skin and airway as well as the pulmonary injuries, particularly if this would require transfer over a long

distance.

Cyanide toxicity is frequently a concomitant exposure with carbon monoxide in enclosed space fires with smoke inhalation. Cyanide exerts its toxic effects through binding to cytochrome c oxidase resulting in cellular hypoxia and usually profound lactic acidosis. Patients may present much like those with carbon monoxide poisoning with headache, nausea, altered mental status, and coma, but without a confirmatory test, this toxin often goes overlooked. With the introduction of hydroxocobalamin, the empiric treatment of severe smoke inhalation patients should be considered as there are few complications with this therapy and great potential benefit.

## KEY POINTS

- Do not wait for obvious signs of airway obstruction in patients exposed to fires in enclosed spaces who display signs of smoke inhalation. The airway can obstruct very quickly, and at that point, endotracheal intubation may be nearly impossible.
- Smoke inhalation patients with altered mental status and acidosis should be considered for cyanide poisoning with early, empirical treatment with hydroxocobalamin.
- Pulse oximetry will be a poor indicator of carbon monoxide levels, and at the time of ED presentation, the venous or arterial carboxyhemoglobin level will have a poor correlation with the level of symptoms or degree of toxicity.
- Emergency hyperbaric oxygen therapy is relatively limited in availability and thus may require long transports. The priorities in smoke inhalation patients are airway management, prevention of hypoxia, and treatment of thermal burns. Patients should be treated with hyperbaric oxygen if stable and if available within a reasonable distance.

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## CO POISONING: IT TAKES MORE THAN O<sub>2</sub>

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**BRYAN WILSON, MD AND CHRISTOPHER G. WILLIAMS, MD, FAWM**

Carbon monoxide is a colorless, odorless gas that is formed by the incomplete combustion of carbon containing materials and that causes a wide variety of nonspecific symptoms. While typical symptoms include headache, dizziness, and nausea, there are reports of symptoms as varied as vomiting, shortness of breath, and even fever with diarrhea. An exposure history such as the use of a combustible fuel for heat in the winter or the use of an indoor generator in an emergency can draw attention to the possibility of carbon monoxide poisoning. A wide range of exposures including riding in a truck, operating a natural gas forklift, swimming behind a boat, and hookah use are more difficult to identify. The presence of characteristic physical exam findings such as cherry-red skin, retinal flame hemorrhages, and cutaneous bullae is uncommon and should not be relied upon. Despite being one of the most common toxic exposures in industrialized countries and causing hundreds of deaths per year, the diagnosis of carbon monoxide poisoning can easily go unrecognized and requires a high index of suspicion. Suspicion of carbon monoxide poisoning can be confirmed by laboratory detection of carboxyhemoglobin (HbCO) or dissolved serum carbon monoxide.

The toxicity of carbon monoxide results from several pathophysiologic effects. The first is impairment of oxygen delivery by preferential binding to hemoglobin with an affinity over 200 times greater than that of oxygen. The effect of this hypoxemia is worsened by a concurrent left shift in the oxygen-hemoglobin dissociation curve. Animal evidence strongly suggests that poisoning heavily relies on direct end-organ damage via inactivation of mitochondrial cytochrome oxidase and the resulting metabolic stress and a



subsequent free radical-mediated inflammatory cascade. Additionally, animal evidence suggests a neuroexcitatory effect of carbon monoxide, which likely enhances the metabolic stress via increased demand. The typical result of these pathways is neuronal cell death, myocardial dysfunction, and long-term development of neuropsychiatric sequelae. Due to the need for diffusion into end organs to mediate toxicity, dissolved serum carbon monoxide levels are likely more predictive of prognosis than are carboxyhemoglobin saturations. Children and fetuses are more susceptible to carbon monoxide poisoning. Previously, this was thought to be due to an increased affinity to fetal hemoglobin though emerging research indicates this may be more attributable to a high metabolic and respiratory demand in the setting of hypoxemia.

Administration of supplemental oxygen is the foundation of carbon monoxide poisoning treatment as it helps to improve hypoxemia and speed elimination of carbon monoxide. On room air, carboxyhemoglobin has a half-life of ~200 minutes. High  $\text{FiO}_2$  via mask can decrease this to 75 minutes, while administration of 100% oxygen at 3 atmospheres of pressure reduces it to 15 minutes. Some evidence suggests that hyperbaric oxygen may confer additional benefit by more promptly restoring cytochrome oxidase activity and minimizing the subsequent inflammatory process. While there is currently insufficient evidence to strongly recommend for or against the administration of hyperbaric oxygen, it should be considered in severe poisonings. In particular, hyperbaric oxygen is frequently recommended for patients with neurologic deficits, syncope, fetal distress, or carboxyhemoglobin >10% in the setting of pregnancy. It is important to weigh the risk of transporting a critically ill patient against the possible benefits of hyperbaric oxygen.

Long-term considerations for these cases revolve around good neuropsychiatric follow-up and source control. It is important to consider sequelae and concurrent exposures when evaluating a patient with carbon monoxide poisoning. Of particular note would be exposure to airway burns or cyanide poisoning in the setting of a house fire. Cyanide is released through the combustion of various plastic products. A serum lactate >10 mmol/L in the setting of smoke inhalation is strongly suggestive of cyanide toxicity with 6 mmol/L often considered confirmatory in cases with a high pretest probability. In the setting of concurrent carbon monoxide and cyanide poisoning, hydroxocobalamin and sodium thiosulfate would be preferable to the methemoglobinemia producing amyl nitrite and sodium nitrite. Other sequelae include pulmonary edema, myonecrosis, compartment syndrome, and acute kidney injury, and the patient should be closely monitored for development of these complications.

Many patients develop a varied range of neuropsychiatric complications such as impaired cognition, mood abnormalities, and abnormal movements in the days to weeks following a poisoning. It is important to ensure the patient has good follow-up to monitor and manage these symptoms should they occur. In addition, all reasonable efforts should be made to identify the source of the exposure and have it contained. The importance of doing this is underscored by high-profile cases such as the fatal carbon monoxide poisoning of a young boy just 2 months after the fatal poisoning of an elderly couple in the same hotel room.

## KEY POINTS

- Consider carbon monoxide poisoning in vague cases that “just don’t make sense.”
- Pediatric and fetal patients are particularly sensitive to carbon monoxide.
- Consider hyperbaric oxygen for severe cases with neurologic manifestations.
- Consider coexposures such as cyanide and sequelae such as hypoxic insult or airway burns.
- Attempt to identify and report the source of the exposure.

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## A RASH THAT IS MORE THAN “JUST A RASH”

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NASH WHITAKER, MD

Few things are more frustrating to emergency physicians (EP) than an unexplainable rash. EP search for that one piece of history that will reveal the diagnosis: “A new detergent? How about a pet? Perhaps new jewelry? Oh, your child just had a cold?” Typical discharge instructions end with a similar unfulfilling undertone—“It’s probably just a virus, it will go away.” Often, this is true; however, missing a more serious pathology when presented with a rash carries significant mortality and morbidity, as is the case with tick-borne diseases.

Ticks are one of the most common vectors for zoonotic disease worldwide. Diseases native to the United States include, but are not limited to, Lyme disease, Rocky Mountain spotted fever (RMSF), tularemia, ehrlichiosis, babesiosis, Q fever, Colorado tick fever, relapsing fever, and anaplasmosis. It is important to recognize that tick-borne illnesses have a geographic distribution and seasonal variation, being more common during summer months when ticks are more active.

The majority of the tick-borne illnesses present initially similar to a viral syndrome: fever, chills, malaise, myalgia, headache, and gastrointestinal symptoms (nausea, vomiting, diarrhea, anorexia) are the typical compilations in the acute phase.<sup>1</sup> These subtle and ambiguous symptoms are seen daily in emergency departments nationwide, making the diagnosis of a tick-borne disease very challenging. Since the majority of patients never recall a tick bite, EP must keep tick-borne disease in their differential diagnosis in all patients presenting with a febrile rash.<sup>2</sup> Of all tick-borne diseases, the three most unique, “can’t miss” diagnoses for the EP are Lyme disease, RMSF,

and tularemia.

For those patients who do present with an attached tick, it is worth noting that there is almost no risk of transmission if the duration of tick attachment is <72 hours.<sup>2</sup> All patients with suspected tick-borne disease will require follow-up either with their primary care physician or an infectious disease specialist, depending on their level of illness. Antibiotics should not be delayed in the setting of high clinical suspicion with any of these diseases. Admission might be required depending on symptom severity and the ability of the patient to tolerate oral antibiotics.

## LYME DISEASE

Lyme disease, the most common tick-borne disease in North America, is caused by the spirochete *Borrelia burgdorferi*.<sup>1</sup> It is endemically located in the northeastern coastal, mid-Atlantic, and north central states, but has been reported in all contiguous 48 states.

There are three stages of Lyme disease. Erythema migrans (EM), a pathognomonic skin eruption that forms at the site of the tick bite, characterizes the early stage. This distinct, vasculitic, nonpruritic rash is infamous for Lyme disease and typically appears 2 to 20 days after exposure.<sup>3</sup> Classically, it is described as a “bull’s-eye” consisting of a red ring with central clearing. A rash is present in 80% of cases, but only 60% of those who develop rash have the classic bull’s-eye appearance.<sup>3</sup>

Dissemination of the spirochetes leads to the middle and late stages of the disease, which are characterized by a multitude of symptoms. The most commonly described symptoms include a transient migratory polyarthritides, A-V nodal block, pericarditis, meningitis, uveitis, or unilateral/bilateral facial nerve paralysis. Additionally, multiple diffuse EM rashes may occur and aid in the diagnosis.<sup>3</sup>

Lyme disease is a clinical diagnosis for the EP. Treatment includes doxycycline or amoxicillin. Polymerase chain reaction or immunoassay confirmation testing is available.

## ROCKY MOUNTAIN SPOTTED FEVER

RMSF is the most severe of all tick-borne diseases in the United States. It has a mortality rate of ~15% to 20%. *Rickettsia rickettsii* is the pleomorphic, intracellular agent. Five states account for 60% of cases: North Carolina, Missouri, Tennessee, Arkansas, and Oklahoma, but sporadic cases have been

reported in most of the other contiguous states.<sup>4</sup> Two-thirds of the total cases are in children younger than 15 years old.

Approximately 80% of cases develop a rash 2 to 4 days after the onset of fever, which is present in 90% of cases. Compared to EM, the rash associated with RMSF is not pathognomonic.<sup>4</sup> The RMSF rash occurs earlier in children and begins as small blanching erythematous macules on the hands, feet, wrists, and ankles. As thrombocytopenia develops and vasculitis worsens, petechial and hemorrhagic lesions will present.<sup>2</sup> This typically occurs as the rash spreads centrally to the trunk.

The antibiotic of choice is doxycycline even for children as the risk of cosmetically appreciable tooth staining is insignificant for a single course of treatment.<sup>1</sup>

Peripheral blood smear, skin biopsy, and immunoglobulin assays may help confirm the diagnosis.

## TULAREMIA

Thought of commonly as a plague-like disease and a potential biochemical weapon, tularemia is caused by a gram-negative coccobacillus, *Francisella tularensis*, which uses the *Dermacentor* tick, along with lagomorphs and rodents as its vectors. It may also be contracted through direct contact of an open wound with an infected host. Cases have been reported in all states except Hawaii, but are most common in the south central United States.

The clinical presentation depends on the portal of entry. Various forms exist, but the most common presentation (80% of cases) is the ulceroglandular form. This presents as an erythematous, tender papule at the site of the tick bite that ulcerates about 2 days later. The ulcer is very slow healing and usually remains present as the disease progresses. Significant, painful, regional adenopathy develops as a bubo forms. The necrotic, purulent, painful lymph nodes will continue to enlarge until potentially rupturing, increasing the chance of bacteremia and septic shock.<sup>1</sup> Inguinal or femoral adenopathy are the most common sites for tick-borne tularemia.

Untreated cases carry a 25% mortality rate. Treatment includes streptomycin or gentamicin. Culture and enzyme-linked immunosorbent assays (ELISA) will aid in the diagnosis for specialists.

### KEY POINTS

- Tick-borne illness must be considered for all patients with a fever and rash.
- Do not delay antibiotic treatment for confirmation with suspected tick-borne disease.
- Know the geographical distributions and seasonal variations to aid in your diagnosis.
- Do not rely upon a known history of tick exposure to aid in your clinical suspicion.

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# DIVING INJURIES: DON'T MISS THESE SERIOUS INJURIES BECAUSE YOU FAILED TO GET THE HISTORY!

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Diving injuries may have greatly varied presentations that make recognition difficult. All patients should be asked about their recreational activities. History taking is paramount regarding all diving activities, as well as postdiving flying or driving to altitude. The most common types of injuries should be your focus of study: barotrauma, pulmonary overpressurization syndromes, and decompression sickness (DCS).

## BAROTRAUMA

Barotrauma is injury incurred due to an inability to equilibrate pressure. Gas-filled spaces are very susceptible to barotrauma. Barotrauma is a constant hazard to scuba divers, free divers, aviators, and airplane travelers ([Tables 127.1 and 127.2](#)).

TABLE 127.1 BAROTRAUMA OF DESCENT

| <b>BAROTRAUMA</b>            | <b>ETIOLOGY</b>   | <b>SYMPTOMS</b>   | <b>TREATMENT</b>   |
|------------------------------|---|---|--|
| External ear squeeze         | Obstruction by wax, bony growths, or earplugs creates a fixed space   | Tympanic membrane (TM) hemorrhage, perforation, ear pain                                | Ascent, clean canals, pain<br>meds                         |
| Middle ear barotrauma (MEBT) | Eustachian tube (ET) dysfunction with inability to equilibrate;<br><i>most common barotrauma (up to 30% of divers)</i>                | Muffled hearing, ear pain, TM hemorrhage, perforation, vertigo, and rare CN VII palsy   | Decongestants, diving rest until seen by ENT, pain<br>meds |
| Mask squeeze                 | Failure to equalize mask results in negative mask pressure—facial squeeze   | Pain, petechial hemorrhages, and subconjunctival hemorrhages                            | Resolves with time   |
| Sinus squeeze                | Obstructed sinus cavities   | <i>Second most common barotrauma</i> ; sinus pain, bloody nasal discharge on ascent     | Decongestants, NSAIDs, diving rest                         |
| Inner ear barotrauma (IEBT)  | Perforation of round or oval window due to equilibration failure, vigorous Valsalva maneuvers, or attempting to clear ears on descent | Ear pain, nausea, vomiting, extreme dizziness, vertigo; ataxia, hearing loss, nystagmus | STAT ENT evaluation  |

**TABLE 127.2 BAROTRAUMA OF ASCENT**



| <b>BAROTRAUMA</b>          | <b>ETIOLOGY</b>   | <b>SYMPTOMS</b>  | <b>TREATMENT</b>                                 |
|----------------------------|---|--|--|
| Middle ear reverse squeeze | ET dysfunction with inability to equilibrate  | Same as MEBT   | Same as MEBT                                     |
| Barodontalgia              | Cavities, decay or poor fillings pressurize; occurs with diving or airplane travel  | Tooth pain, headache; rare tooth fractures   | Pain meds; dental evaluation                     |
| Alternobaric facial palsy  | ↑ Middle ear pressure compressing CN VII ( <i>ischemic neuropathy</i> ); divers who fly after diving, unpressurized plane passengers, victims of in-flight explosive depression | <i>Unilateral CN VII palsy, ataxia, vertigo, nausea, vomiting</i>                    | Symptoms resolve over time                       |
| Alternobaric vertigo       | Pressure differences between middle ear spaces with asymmetrical vestibular organ stimulation   | Vertigo, nausea, vomiting, transient hearing loss                                    | Abrupt relief with clearing of ears or redescend |
| GI barotrauma              | Gas expansion on ascent; usually from dietary indiscretion or head first water entry  | Nausea, belching, flatulence, stomach pain, reflux, rare gastric rupture, and sepsis | Subsides in time after surfacing                 |

## PULMONARY SYNDROMES

## OVERPRESSURIZATION

These injuries are the result of overpressurization on ascent, leading to barotrauma of the lungs and gas escape. Arterial gas embolism (AGE) is the most lethal injury and second only to drowning as a cause of death in divers. The most common cause is breath holding on ascent ([Table 127.3](#)).

**TABLE 127.3 PULMONARY OVERPRESSURIZATION SYNDROMES**

| BAROTRAUMA                               | ETIOLOGY  | SYMPTOMS   | TREATMENT  |
|--|---|--|--|
| Subcutaneous emphysema                   | Air enters neck tissues   | Chest fullness, voice changes, dysphagia, neck pain, crepitus                                      | High-flow O <sub>2</sub>   |
| Pneumomediastinum, mediastinal emphysema | Air enters mediastinum through perivascular sheath  | CP, SOB, Hamman sign   |  |
| Pneumothorax                             | Air enters pleural space  | CP and SOB, dry hacking cough, dyspnea, shock, cardiac arrest                                      | High-flow O <sub>2</sub> ; needle decompression; tube thoracostomy                             |
| Arterial gas embolism                    | Alveolar rupture into vascular space with gas embolization to the heart, brain, spine, or other tissues | LOC at surface, confusion, paralysis, headache, agitation, vertigo, shock, acute MI, stroke, death | High-flow O <sub>2</sub> ; immediate transport to a HBO <sub>2</sub> chamber for recompression |

## DECOMPRESSION SICKNESS

DCS (“the bends”) is a type of decompression illness (DCI) that presents following deep dives when gas, previously in solution within the plasma, comes out of solution and embolizes downstream. It occurs in divers, saturated with inert gases, due to deep dive profiles. It also occurs in miners and caisson workers. There are three kinds of type I DCS (milder symptoms) and three kinds of type II DCS (severe symptoms with a high risk of death or severe disability). ***All types of DCS mandate treatment with high-flow O<sub>2</sub> at once, followed by transfer to a hyperbaric chamber for immediate recompression therapy (Tables 127.4 and 127.5).***

**TABLE 127.4 TYPE I DECOMPRESSION SICKNESS**

| TYPE OF DCS                | SYMPTOMS  |
|----------------------------|---|
| Musculoskeletal DCS        | Gradual onset of dull aching joint pain (usually shoulder or elbow) usually occurring within 24 hours of surfacing          |
| Cutaneous DCS “skin bends” | Pruritus and formication-like symptoms or a mottled rash often termed “cutis marmorata” or a ring like “peau d’orange” area |
| Lymphatic DCS              | Uncommon form of DCS with swelling of the soft tissues distal to the lymph nodes and resembling lymphedema                  |

**TABLE 127.5 TYPE II DECOMPRESSION SICKNESS**

|  |  |
|--|--|
| Neurologic DCS<br>60%–70% of type II DCS | <i>Spinal cord most often affected</i> ; often occurs in recreational divers; with progressive numbness and paresthesias; abdominal pain; motor weakness with bowel and/or bladder incontinence; <i>significant cognitive impairment</i> |
| Pulmonary DCS<br>“the chokes”            | <i>Persistent, dry cough and inspiratory CP</i> ; seen more often with high-altitude DCS, caisson workers, and tunnelers   |
| Vestibular DCS<br>“the staggers”         | Dizziness, nausea and vomiting, hearing loss, tinnitus, nystagmus; <i>confused with MEBT and rare in recreational divers</i>   |

## KEY POINTS

- For any **possible** diving-related accident, call the **Diver’s Alert Network** immediately (919-684-9111). A diving medical specialist will guide you to the best course of action.
- Always consider DCI (either DCS or AGE) as the cause of any injury occurring during or after a dive. **Have a very low threshold in considering these diagnoses.**
- Take a comprehensive diving history and ask them to show you their dive computer.
- Abdominal pain may be an early sign of spinal cord DCS.
- **A detailed neurologic exam is paramount**, especially cerebellar, sensory, and cognitive functioning.

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# SECTION VIII

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## HEENT

# GIANT CELL ARTERITIS: WHO THE HECK IS HORTON AND WHY SHOULD I WORRY ABOUT HIS HEADACHE?

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AISHA PARKER, MD AND JAMES AIKEN, MD, MHA

## BACKGROUND

Horton disease, temporal arteritis, cranial arteritis, and arteritis of the aged are all synonyms for the disease known commonly as giant cell arteritis (GCA). GCA is the most common manifestation of systemic vasculitis in adults. The earliest description of this disease dates to 1350 BC with a drawing on the tomb of Pharaoh Pa-Aton-Em-Heb.<sup>1</sup> The relief shows an elderly blind harpist with an enlarged and discontinuous temporal artery.

GCA is a systemic necrotizing vasculitis that affects medium and large vessels causing granulomatous inflammation. This disease has a predilection for the superficial temporal, ophthalmic, proximal vertebral, and posterior ciliary arteries usually sparing intracranial arteries but can affect arteries of all sizes and locations. Of note, there are three clinical subtypes of the disease:

- *Cranial arteritis* (temporal arteritis) presents with headache and jaw claudication and may progress to blindness. It differs from the other subtypes, in that it involves carotid artery branches only.
- *Systemic inflammatory syndrome* presents with fatigue, myalgias, weakness, night sweats, and fever of unknown origin. Symptoms

overlap with polymyalgia rheumatica (PMR).

- *Large vessel vasculitis* affects the subclavian arteries, axillary arteries, and aorta. It presents with arm claudication and arterial bruits and may progress to aortic dilatation and aneurysm. Patients with large vessel vasculitis have a negative ESR.

Although rare with a reported prevalence of 0.5 to 27 cases per 100,000 in people aged 50 or older, GCA is associated with significant morbidity, especially if not treated immediately,<sup>1</sup> and so must not be overlooked. Sequelae may include blindness, renal artery stenosis, aortic dissection, and ischemic stroke. GCA affects women at a rate threefold higher than men with a higher overall incidence in Scandinavian peoples. Of note, there seems to be a genetic association with PMR; roughly 50% of patients with PMR are also diagnosed with GCA.

## PRESENTATION

Patients diagnosed with GCA are classically in the seventh to eighth decade of life; however, one should maintain a high clinical suspicion in patients older than age 50. Seventy-two percent of patients complain of new-onset headache or change in their chronic headache pattern, which can be associated with scalp tenderness around the temporal and occipital arteries.<sup>2</sup>

The most reliable clinical sign is jaw claudication<sup>3</sup> (likelihood ratio [LR] of 4.2). In fact, jaw claudication is pathognomonic for GCA and is caused by ischemia of the masseter muscle. Other associated signs include a palpable prominent temporal artery (LR 4.6) with segmental beading (LR 4.3) and diplopia (LR 3.4). Ocular symptoms such as diplopia, amaurosis fugax, and ptosis are due to vascular insufficiency. If the patient is found to have central retinal artery occlusion, fluorescein angiography is needed to detect concomitant posterior ciliary artery occlusion.<sup>4</sup>

## DIAGNOSIS

For the emergency physician, consideration of GCA is the most important part of disease treatment; one cannot effectively intervene if one does not consider the diagnosis.

Diagnosis is made with three of the five following criteria per the American College of Rheumatology<sup>5</sup>:

- 1) Age of onset 50 or older

- 2) New onset of headache
- 3) Clinical temporal artery abnormality
- 4) ESR > 50 mm/hour
- 5) Abnormal temporal artery biopsy

Other notable diagnostic factors:

- If CRP is > 2.45 mg/dL, odds of + biopsy is 5× greater.
- If platelets are > 400,000, odds of + biopsy is 4× greater.
- If ESR is 47 to 107 mm/hour, odds of + biopsy is 1.5× greater.
- ESR should be age adjusted: normal is an ESR less than age/2 for men and (age + 10)/2 for women.

Negative biopsy does not exclude GCA as the lesions are not contiguous. Nonetheless, bilateral temporal artery biopsy is the gold standard for diagnosis. Emergency physicians should refer patients for biopsy within 48 hours of initiation of steroid therapy. After 1 week of treatment, only 50% of biopsies will be positive. Therefore, a delay in biopsy may result in false-negative results. If the biopsy is negative, imaging may aid in definitive diagnosis to justify chronic steroid treatment. Color duplex ultrasonography of the temporal artery is useful in ruling in GCS with a specificity of 80% to 100% in the presence of a halo sign. However, its sensitivity is low especially in the early phases of inflammation. PET and MRI are also a useful imaging modality in large vessel vasculitis subtype of GCA.<sup>1</sup>

## TREATMENT

Steroid treatment (prednisone 40 to 60 mg/day) should be initiated in the ED prior to biopsy if the diagnosis is likely. This may not restore preexisting vision loss but can prevent further damage. While most patients can be discharged home with close follow-up, patients with severe disease or visual loss should be hospitalized for high-dose steroids. It is imperative to discuss the importance of medication compliance in the discharge instructions. Other targeted immune modulators can be also be employed such as tocilizumab; however, efficacy has yet to be established and prednisone continues to be the mainstay of treatment. Patients are typically treated with a slow steroid taper over the course of 1 to 2 years in order to prevent relapse.

## KEY POINTS



- Consider the likelihood of GCA in all new-onset headache in patients over 50 years.
- GCA is a disease of clinical diagnosis and a medical emergency.
- Give steroids immediately if the symptoms indicate a high likelihood of the disease.
- Neither a normal ESR nor a negative biopsy rule out GCA.
- Patients will need referral to surgery (general, plastics, or ophthalmology) for biopsy and rheumatology for continued care.

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# SIGHT-THREATENING ZOSTER OPHTHALMICUS: HOW TO RECOGNIZE AND TREAT

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## BACKGROUND

Herpes zoster ophthalmicus (HZO) is shingles in the V1 distribution of the trigeminal nerve with ocular involvement. Rapid recognition and treatment are critical due to potential loss of vision.<sup>1</sup> Over half of patients with zoster in V1 will have ocular involvement.<sup>2</sup>

## PRESENTATION

The typical patient will be elderly or immunocompromised (HIV/AIDS, poor nutrition, organ transplant, etc.). Chronologically, the typical story will begin with a weeklong history of prodromal flulike symptoms, followed by a more recent unilateral vesicular rash with crusting lesions and pain in the V1 distribution, obeying the midline. If the rash involves the tip of the nose (Hutchinson sign), it is highly specific for ocular involvement. This is because the nasociliary branch of V1 innervating the tip of the nose is the same branch that innervates the globe.<sup>2</sup> Ocular symptoms can range from tearing, redness, photophobia, visual disturbances to lid droop. Ocular involvement may be delayed with respect to the onset of the rash, ranging from no delay to years; however, most ocular involvement presents within 2 weeks of the rash.<sup>3</sup>

Unfortunately, not all of your patients come packaged with the helpful

trademark rash. A minority will present with ocular involvement only.<sup>3</sup> Patients with HIV/AIDS can have a generalized rash and appear more severely ill.

Any part of the eye can be involved: eyelid, conjunctiva, sclera, cornea, anterior chamber, iris, retina, and cranial nerves. The majority (2/3) will develop corneal involvement.<sup>2,4</sup> Areas for potential visual morbidity include the cornea due to corneal thinning and perforation, the anterior chamber due to uveitis with subsequent glaucoma and cataract formation, and the retina due to retinal detachment and necrosis.<sup>3</sup>

Varicella-zoster is considered the primary cause of acute retinal necrosis (ARN) and progressive outer retinal necrosis (PORN). PORN is a severe version of ARN, usually found in patients with AIDS.<sup>3</sup> ARN will present as blurred vision and pain. Rapid loss of vision ensues due to retinal detachment, unfortunately a common complication of ARN.<sup>1,5</sup> Nearly half of ARN cases will present bilaterally.<sup>6</sup>

## DIFFERENTIAL

Conjunctivitis: viral (HSV, adenovirus), bacterial (gonorrhea, *Chlamydia*), allergic, and parasitic (acanthamoebic keratitis in contact lens wearers)

## TESTS

Visual acuity, fluorescein staining, slit lamp exam, and intraocular pressures

If you are concerned for HZO, slit-lamp exam is a must to check for corneal involvement.

HZO findings include:

- 1) Punctate or pseudodendrite lesions (vs. HSV keratitis: “true” dendrite)
- 2) Cell and flare

Pseudodendrites (HZO) versus dendrites (HSV keratitis): pseudodendrites stain minimally, have a “stuck on” or elevated appearance, and lack terminal end bulbs, while true dendrites stain brightly, ulcerate, and have terminal end bulbs.<sup>7</sup> Decreased corneal sensation is suspicious for HSV and acanthamoebic keratitis (try a fine cotton wisp of a cotton tip).<sup>8</sup>

## TREATMENT

- 1) Skin, if lesions are <7 days old:  
Acyclovir 800 mg PO 5×/day OR famciclovir 500 mg TID OR valacyclovir 1,000 mg TID ×7 to 10 days.<sup>9</sup> Studies report pain relief and decreased likelihood of developing ocular complications with oral acyclovir if given within 72 hours of onset.<sup>10</sup> Realize 5×/day can be difficult to remember, so valacyclovir may be preferred for some.
- 2) Skin lesions are susceptible to secondary staph/strep: erythromycin 2% ointment and warm compresses.<sup>9</sup>
- 3) Erythromycin 0.5% ophthalmic ointment BID.<sup>9</sup>
- 4) Iritis (suggested by painful photophobia): prednisolone acetate 1% drop q1–6h, BUT BEWARE; using topical steroids in HSV keratitis is contraindicated; this is why slit lamp and ophthalmology consult are important.<sup>9</sup>
- 5) Pain: oral opioid analgesia, cycloplegic agents (cyclopentolate 1% one drop TID), and cool compresses.<sup>9</sup>
- 6) Severe cases (HIV/AIDS, ARN/PORN): admission and IV acyclovir. ARN/PORN requires over 3 months of both PO and IV acyclovir along with corticosteroids.<sup>3</sup>
- 7) Patients <40 years old need outpatient workup for immunosuppression.<sup>9</sup>
- 8) Mild cases: follow up outpatient with ophthalmology within 2 days.
- 9) Postherpetic neuralgia responds to amitriptyline 25 mg PO QHS.<sup>8</sup>

## KEY POINTS

- Over half of patients with zoster in V1 will have ocular involvement.<sup>2</sup>
- Treatment initiation within 72 hours = symptomatic relief and prevention of visual morbidity
- Tip of the nose involvement = Hutchinson sign = ocular involvement
- DO NOT use steroid drops in HSV keratitis. Ophthalmology should be consulted before steroid use.

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## AND THE EYES HAVE IT

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**SUMMER STEARS-ELLIS, MD**

The term optic neuritis (ON) refers to a spectrum of diseases that cause inflammation of the optic nerve. It usually presents as a triad of subacute unilateral loss of vision, periocular pain and impaired color vision in an otherwise healthy young adult. ON is thought to be mostly idiopathic, though it can be related to demyelinating diseases (MS being the most common) and other less common etiologies such as autoimmune disease (SLE, sarcoidosis, Sjögren syndrome, Behçet disease), inflammatory/postvaccination immunological responses, or infectious/parainfectious processes (herpes zoster, measles, West Nile, adenovirus, syphilis). ON is also seen more prevalently in Caucasians from higher latitudes and rarely in African Americans and Asians. However, there is some literature that supports the theory that individuals that migrate to areas of higher incidences of ON/MS before puberty take on the same rate of incidence of ON/MS in that area.

A variety of serious disease processes can mimic ON, therefore, it is vital that you be able to distinguish between a typical clinical presentation of ON and atypical signs of ON as your differential diagnosis, laboratory/imaging studies, and potential treatments will differ.

Demographics and symptoms of ON include age < 50 (usually peaks between 15 and 49), female > male (3:1), acute to subacute onset (hours to weeks), previous history of ON or MS, and periocular pain especially with eye movement (90%). In adults, a unilateral loss of visual acuity is typically present although its degree may be variable (20/20 in 10.5% vs. no light perception in 3.1%). In children, it may be bilateral, such as following an infection. The optic nerve head is normal in adults (65%) but can be swollen in children (35%). Typically, ON will show spontaneous visual improvement after 2 to 3 weeks (>90%), no deterioration in vision after appropriately prescribed steroid treatment and impaired color vision.

Atypical demographics and symptoms of ON include age >50 or <12, simultaneous or sequential bilateral ON, severe visual loss (no light perception) which progresses >2 weeks from symptom onset, painless/painful/persistent pain >2 weeks, abnormal ocular findings, no visual recovery/continued worsening symptoms, systemic diseases other than MS, visual deterioration after appropriately prescribed steroid treatment, previous history of neoplasia and optic atrophy with no history of ON or MS.

Atypical signs of ON should prompt the provider to widen their differential diagnosis and alter their investigation and treatment plans. This should include mimickers of ON such as optic neuropathies (AION, LHON), toxic or metabolic causes (medications, carbon monoxide, methanol and other alcohols, chemotherapy agents, nutritional/vitamin deficiencies [B<sub>12</sub>]), as well as compressive optic neuropathies (aneurysms, tumors, cancers, mass lesions, thyroid eye disease, and other orbital abnormalities).

The examination of a patient suspected of having typical ON should include a slit-lamp examination color vision assessment, visual acuity, peripheral visual field determination, and fundoscopic exam. These can help exclude an ocular cause of visual loss. Neurological examinations as well as an orbital and brain MRI (with/without gadolinium) should be performed within 2 weeks of symptom presentation as this will not only help confirm the diagnosis, but in the case of suspected MS, an MRI with gadolinium can be used as a predictor for developing clinically definite MS. Laboratory studies and lumbar puncture are not recommended by the Optic Neuritis Treatment Trial (ONTT) in patients with typical symptoms of ON.

In patients with atypical symptoms of ON, the above examinations should be performed. In addition, an orbital and brain MRI with gadolinium are mandatory, but if not available, a CT scan with contrast should be performed. Laboratory tests and lumbar puncture with CSF analysis can help to determine infectious causes, optic neuropathies, inflammation or vasculitis. Baseline labs you may consider ordering could include a CBC, CMP, ESR, CRP, ANA, B<sub>12</sub>, RF, virology, syphilis, HIV, and various other studies depending on your clinical suspicion and likely in consultation with ophthalmology.

The importance of being able to determine typical ON from any other insidious cause of vision loss is vital toward helping your patient receive the appropriate diagnosis, medical treatment and follow-up. As many patients that will develop MS have ON as a presenting symptom, your immediate actions and appropriate interventions are important to their speedy recovery.

## KEY POINTS

- ON includes a spectrum of diseases that cause inflammation of the optic nerve.
- Typical ON needs to be distinguished from signs of atypical ON as the etiology, workup, and treatment will differ.
- Laboratory studies and lumbar puncture are not recommended for typical ON.
- Any atypical symptoms should include laboratory studies, radiographic studies, and likely lumbar puncture if clinical suspicion warrants.

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## “YOUR PATIENT HAS A RETROBULBAR HEMATOMA. I THINK HE’S GOING TO NEED A CANTHOTOMY.”

JONATHAN DANGERS, MD, MPH

Sitting in a bar with residents after a shift, when I get the above text message from the night doc, I had checked out my patients to an hour earlier. It was referring to a “wake and shake” drunk patient who had been assaulted and CT head pending at the time of checkout. Moments later: sweaty palms, sinking feeling, muttering expletives, and no longer enjoying my beer. True story. To avoid a similar text, read on.

Orbital compartment syndrome occurs from bleeding or swelling in the partially closed orbital space. Pressure rises rapidly, pushes the globe forward putting tension on the optic nerve, and compresses the vasculature, venous first.<sup>1</sup> Loss of vision may be rapid and permanent. You can prevent that.<sup>2</sup>

### CAUSES

The vast majority will be caused by facial trauma, but nontraumatic causes are many.<sup>1</sup> Most atraumatic retro-orbital hematomas occur with:

- 1) **Tendency to bleed:** coagulopathies (inherited, acquired), anticoagulants, and thrombolytics
- 2) **Postsurgical:** orbital or periorbital (ocular, extraocular, sinus) and



intracranial

- 3) **Increased pressure:** Valsalva, childbirth (both mother, child), and sinus disease

Diagnosis and treatment of traumatic and atraumatic etiologies are identical.

## CLUES

- 1) **Painful “tense” proptosis:** Easy to dismiss as eyelid swelling when swollen shut.
- 2) **Decreased visual acuity:** Determine best possible and reevaluate frequently.  
Eye chart → count fingers → describe shapes → movement → light perception.
- 3) **Pupillary response:** Sluggish response to light compared to unaffected eye; afferent pupillary defect.
- 4) **Restricted or painful extraocular movements.**
- 5) **Elevated intraocular pressure (IOP):** Check several measurements, compare to the unaffected eye, and reevaluate. Don't jab too aggressively as this may falsely elevate the Tono-Pen reading. Don't measure if you suspect globe rupture.

Normal patients will tell you the diagnosis. This isn't about those patients. Real patients are often uncooperative, intoxicated, altered, or intubated. These patients are high risk for a miss and need your attention. Don't dismiss proptosis as periorbital swelling. Pry the swollen lids open. Use the Tono-Pen. Flip the room lights off and accurately check the pupillary response. Remember to perform serial exams as changes may occur quickly.

Retro-orbital hematoma is a clinical diagnosis. Point of care ultrasound or CT can help confirm but may delay care. Neither is necessary for diagnosis and neither will definitively tell whether to intervene or when not to.

## WHEN TO INTERVENE

The differential diagnosis is broad for patients with proptosis *or* decreased vision *or* elevated IOP but reasonably specific for orbital compartment syndrome when all three are present. Indication to cut is the presence of all three:

**Proptosis.**

**Decreased vision.** Your best judgment (see above).

**IOP > 40.** (Some say >30.)

Canthotomy should be performed ***within 60 to 90 minutes of injury*** to maximize the chance of full recovery.<sup>2</sup>

## HOW TO MAKE THE CUT

Retro-orbital hematoma is a true emergency, and lateral canthotomy is a sight-saving procedure with all the potential for greatness or failure. Deferring to an experienced specialist (ophthalmology) is prudent and reasonable, but not always possible. There are few opportunities to perform or practice canthotomy clinically, so make the most of every opportunity: video reviews, cadaver practice, and observing other providers. When the time comes, you can be hesitant and avoidant or confident and ready to act. It all depends on preparation.

The lateral canthal ligaments attach the lids to the orbital rim, prevent forward displacement of the globe, and must be severed to relieve orbital pressure.

- 1) Anesthetize the lateral lid using lidocaine with epinephrine. Aim the needle laterally to avoid globe injury.
- 2) Clamp and crush the canthus for 60 seconds to decrease bleeding.
- 3) Use forceps to pull the lid away from the globe. Make a 1-cm horizontal cut with sterile scissors (the lateral canthotomy).
- 4) Find the inferior crus of the lateral canthal ligament. You may have to rely on “feel.” Imagine a taut band that snaps back when you rake the forceps over it. Pull the ligament outward with forceps and cut through (cantholysis). Scissor tips should be pointed inferior and lateral.
- 5) The ligament and lower lid should feel slack once released.
- 6) Recheck IOP and visual acuity.
- 7) Get the patient definitive care in the OR.

## ADJUNCTIVE TREATMENT

Elevate the head of the bed.

Treat pain and nausea.

Apply ice and avoid compression.

Medication recommendations to decrease swelling and IOP are expert opinion only since no reliable clinical trials exist in this setting.<sup>1</sup> Many

sources recommend the following unless contraindicated:<sup>3–5</sup>

- 1) Acetazolamide 500 mg IV bolus
- 2) Mannitol 1 g/kg IV over 30 minutes; contraindicated if hypotensive or a bleeding trauma patient
- 3) Hydrocortisone 100 mg IV

## KEY POINTS

- Recognition is the first step. Consider orbital compartment syndrome in the atraumatic patient.
- Conduct a detailed eye exam in every noncommunicative patient with facial trauma.
- Memorize the short checklist of indications for canthotomy and cantholysis.
- Consider adjunctive medications until definitively managed.
- Most importantly, learn the procedure ahead of time so you can act promptly and with confidence when you need to save someone's vision.

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# BEWARE THE SORE THROAT THAT KILLS

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**DIANE RIMPLE, MD, FACEP**

The evolution of epiglottitis from an illness of children to one of adults since the introduction of the HiB vaccine is not news. You might wonder if the adult presentation of the disease is any different from the classic picture of the toxic, drooling, tripodding, *dying* child. In fact, the “new” epiglottitis has a spectrum of presentations from mild illness with sore throat and hoarseness to the more classic and toxic appearance. This makes it that much more difficult to diagnose—it doesn’t follow a tightly worded script. An adult with stridor, drooling, fever, and history of a sore throat may not present a diagnostic conundrum (though they may scare the pants off you), but what about the patient presenting before they are about to lose their airway? Many patients ultimately diagnosed with epiglottitis are not diagnosed on their first visit to a health care provider.

Invasive HiB disease still affects adults and children in the United States though an estimated 75% of HiB infections now involve adults.<sup>1,2</sup> Most of these cases are pneumonia, and many are caused by less invasive, unencapsulated subspecies of HiB. Epiglottitis (+/- abscess) is the most serious form of invasive HiB disease still encountered, but epiglottitis in adults has been found to be due to other bacteria as well—primary streptococcal infections: group A strep, strep pneumonia, and *Haemophilus parainfluenzae*.<sup>3</sup> While mortality has dropped from 7% to 1% in the remaining pediatric cases currently seen, in adults, the mortality rate has remained at 7% for the last two decades.<sup>1,2,4</sup> Morbidity and mortality in epiglottitis are closely related to late (emergent) airway management and may be due to late presentation or delay in diagnosis.<sup>5</sup>

Patients with epiglottitis present with sore throat and pain with swallowing. Most will also have fever. There is a 3:1 male to female ratio, the average age is in the 40s, and smoking seems to predispose to the disease (along with diabetes and other immunocompromised states).<sup>6</sup> The onset in adults tends to be less dramatic than in children. Instead of hours, many adults with epiglottitis have several days of symptoms prior to presenting for care. Interestingly, those who present earlier (within hours) seem to have a more aggressive disease course and are more likely to need intubation.

The differential for a patient with epiglottitis includes upper respiratory infection, viral pharyngitis, strep throat, peritonsillar abscess, retropharyngeal abscess, and esophagitis. A soft tissue lateral neck x-ray is often the initial test if one suspects epiglottitis. While the specificity of x-rays is good, there is a false-negative rate of about 20%.<sup>1</sup> The best way to truly rule in or rule out epiglottitis is direct visualization, such as with a fiberoptic nasopharyngeal scope.

Management of epiglottitis involves early definitive airway management. This is a situation where ego shouldn't get in the way of best care for your patient. Call for help if ENT or anesthesia is available. Many texts recommend awake fiber optic nasopharyngeal intubation if possible. Consider using a bougie to get around an engorged epiglottis if you are performing endotracheal intubation. An extraglottic device may actually make airway compromise worse by pushing the engorged epiglottis over the trachea, and an inflamed epiglottis tends to swell rapidly with even minor physical contact. Always be prepared to perform a cricothyrotomy. Your cric hook should be itching in your pocket while managing these airways.

Medical management includes steroids and broad-spectrum antibiotics like a third-generation cephalosporin. Use of inhaled racemic epinephrine is controversial but hasn't been studied in any recent or adult patient literature. Admission to an ICU for close airway observation of the nonintubated patient is typically warranted.

Finally, epiglottitis can be triggered by caustic inhaled or thermal airway injuries. There have been many reports of epiglottitis caused by inhalation of crack cocaine or steam.

## KEY POINTS

- Hold a high index of suspicion in an adult presenting with sore throat and normal oropharynx.

- Stridor is always a very bad sign.
- Getting an x-ray isn't good enough.
- Maintain a low threshold to just take a look—direct visualization with a nasopharyngeal scope is relatively quick and easy.
- Call for help, call early.

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# **CONSIDER A DEEP SPACE NECK INFECTION IN A CHILD WITH FEVER AND NECK PAIN OR TORTICOLLIS**

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**JOANNA SCHWARTZ, BA, MD**

Sore throat or neck pain is a common reason for children to come to the emergency department (ED). The majority of the time children will have a benign and self-limiting illness. Occasionally, however, a life-threatening deep space neck infection can be present. Subtle clues and a high level of suspicion can help the ED provider pick out which children are truly ill. The two most common types of deep neck infections are retropharyngeal (RPA) and parapharyngeal abscesses (PPA), which occur either by direct penetrating trauma or through spread from a contiguous area. They are typically uncommon complications of upper respiratory infections in children resulting from spread of infection to an eventual suppuration of retropharyngeal lymph nodes. Similarly, the majority of parapharyngeal infections arise from the lymph nodes within the parapharyngeal space. Pediatric deep space neck infections have historically been associated with significant morbidity and mortality due to airway obstruction, mediastinitis, jugular vein thrombosis, aspiration pneumonia, or aneurysm of the carotid artery. Advances in early detection through imaging and antibiotic treatment have greatly reduced these complications and now deep space neck infections seldom lead to long-term consequences if detected early.

RPA is more common in boys and the mean age at diagnosis is 4 years old. The retropharyngeal lymph nodes are thought to involute around 5 years

old, making an abscess an uncommon finding in an older child unless related to trauma. The most common symptoms at presentation include fever, neck pain, torticollis, dysphagia, neck mass, sore throat, and, less commonly, respiratory distress and/or stridor. The physical examination frequently reveals restricted neck mobility and cervical lymphadenopathy. RPA often presents in preverbal children, so it can be hard to get a good symptom history. As a result, restricted neck mobility in a child with fever is the sentinel clinical clue to a diagnosis of RPA or PPA. Be cautious of attributing a child who does not want to move her neck to a garden-variety pharyngitis or torticollis.

The rather broad differential diagnosis includes pharyngitis, stomatitis, cervical lymphadenitis, meningitis, and, in cases of respiratory distress or stridor, croup and epiglottitis. Peritonsillar abscess, though not technically a deep space neck infection, can present with many similar symptoms. However, a bulge in the posterior aspect of the soft palate and uvular deviation should be readily apparent on physical exam.

An adequate examination of the oropharynx can be difficult in children and often may be normal in a child with a deep space neck infection. In fact, a normal pharynx in a symptomatic child should be a red flag. If a child is cooperative, a posterior pharyngeal bulge may be seen. A lateral neck radiograph may aid in the diagnosis of RPA. However, it must be a true lateral and the child must keep the neck in extension during inspiration to avoid a false thickening of the retropharyngeal space. As can be imagined, this is often challenging even in pediatric specialty centers, and false-positive imaging studies are common. Findings consistent with an RPA on plain films include a prevertebral space that is increased in depth compared with the anteroposterior measurement of the adjacent vertebral body or a retropharyngeal space that is  $>7$  mm at C2 or 14 mm at C6. Another method is that the width of the prevertebral space should measure no more than half the thickness of the vertebral body from C1 to C4 or the full thickness from C5 to C7. The prevertebral space can change with crying, particularly in infants, swallowing, and ventilation, so it is generally not the recommended radiologic study of choice unless the patient is unstable.

CT of the neck with contrast is the imaging modality of choice for RPA and PPA; the sensitivity for detecting an abscess ranges from 72% to 81% and specificity is 57% to 59%. When an abnormality is detected on CT scan, it is important to consult with an ENT specialist to help guide management. Surgical drainage used to be considered the standard treatment for RPA or PPA. Current literature now supports an initial trial of intravenous antibiotics unless the patient is unstable or there is an abscess with rim enhancement with an axis  $>20$  mm on CT scan. Benefits of medical management include



avoidance of iatrogenic injury to cranial nerves or great vessels while potentially not increasing the duration of hospitalization.

The cultures from abscess drainage are often polymicrobial. The organisms most commonly recovered from intraoperative cultures are *Staph aureus*, including MRSA, *Strep pyogenes* (group A streptococcus), and respiratory anaerobes. Initial antibiotic therapy should target these organisms.

Whether treated medically or surgically, duration of hospital stay for both groups averages 3 to 5 days for pediatric deep space neck infections. Relapse rates are low, below 5%. When detected early and appropriately treated, children with deep space infections typically have excellent outcomes.

## KEY POINTS

- Consider a deep space neck infection in a child with fever and limited neck mobility or torticollis.
- Lateral neck radiographs can be useful in aiding the diagnosis if properly performed, particularly in an unstable patient.
- CT of the neck with contrast is the imaging modality of choice, though the specificity for abscess can be as low as 57%.
- Early consultation with an ENT specialist is essential to plan treatment.
- In a stable child with an abscess <20 mm on CT, an initial trial of IV antibiotics is the current treatment recommendation, with subsequent surgical intervention if no improvement.

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## LEMIERRE SYNDROME: A ROYAL PAIN IN THE NECK

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FRANK J. EDWARDS, MD, FACEP

Though sore throats come in many varieties, our pharyngitis patients are typically young and healthy and will recover regardless of whether we prescribe antibiotics or not. For good reason, we are trying to get away from overprescribing antimicrobials for common infections. There is even a school of thought that treating group A beta hemolytic streptococcal pharyngitis with antibiotics may only shorten the disease course by an insignificant increment and yet not prevent rheumatic fever or poststreptococcal glomerulonephritis. However, a less commonly known complication of pharyngitis—Lemierre syndrome—could be enjoying a resurgence in frequency.

In 1936, Andre Lemierre described a series of young adults who developed a bizarre constellation of symptoms in the days and weeks following an acute episode of pharyngitis. The four classic characteristics of Lemierre syndrome, also known as “postanginal sepsis” (from angina, sense of constriction or pain), are (1) history of a recent sore throat, (2) positive blood cultures, (3) clinical or radiographic evidence of thrombophlebitis of the internal jugular vein, and (4) metastatic spread of infection to remote sites, most commonly the lung. Positive blood cultures for *Fusobacterium necrophorum*, the usual culprit, are highly characteristic.

*Fusobacterium necrophorum* is a pleomorphic, anaerobic gram-negative rod and part of the normal flora of the human oral cavity. Under proper circumstances following a pharyngeal infection, *F. necrophorum* becomes pathogenic, taking advantage of localized tissue destruction and anaerobic conditions in the lateral pharyngeal region. Due to the close proximity of the internal jugular vein, septic thrombophlebitis can develop and seed distant

structures, such as the lungs, with septic emboli. Carotid arteritis may also develop.

In the emergency department (ED), making the diagnosis is the primary challenge.

The median age of patients with Lemierre syndrome is 19. They may or may not appear toxic, but fever is usually present, as is headache, along with a lingering sore throat. The sore throat may have been present from several days to several weeks and may be improving. Unilateral neck pain and accompanying swelling are frequently seen. Other symptoms depend upon the location of metastatic infection, which can range far and wide. The lungs are frequently involved, causing pneumonia. Infection and inflammation may also spread directly up the carotid sheath to involve the cavernous sinus, inducing the dreaded complication of cavernous sinus thrombosis. When this occurs, there may be dysfunction of the cranial nerves (CN) that pass through this region: CNs VI, IX, XI, and XII. With cranial nerve involvement, the literature reveals several cases in which presenting symptoms include diplopia secondary to paralysis of the lateral rectus muscle due to involvement of CN VI, the abducens nerve. Because the cervical sympathetic chain travels through the cavernous sinus, Horner syndrome (ptosis, miosis, and anhidrosis) has also been reported with cavernous sinus involvement.

Lemierre syndrome usually begins less than a week after the onset of pharyngitis and is often missed in its early stages. The alert practitioner needs to keep a high index of suspicion when evaluating an ill-appearing adolescent or young adult that presents with a long-lasting sore throat with unilateral neck pain and signs of a significant infection elsewhere, particularly the lungs.

Blood cultures will be positive for *Fusobacterium* in up to 50% of cases. *Fusobacterium* is a fastidious bug that can take many days to grow out. Keep a low threshold for imaging the neck to look for jugular vein or carotid artery intraluminal filling defects. Contrast-enhanced CT is useful, but bedside ultrasound may quickly reveal the diagnosis. A chest x-ray should be performed to look for pneumonia, often bilateral due to hematogenous seeding. Other sites of metastatic infection include long bones, joints, liver, brain, meninges, endocardium, mastoid, and facial sinuses.

Interestingly, the initial throat infection does not have to be bacterial. It is not *F. necrophorum* that causes the pharyngitis; this organism invades the tissue opportunistically due to localized hypoxia. As many as 10% of cases of Lemierre syndrome may follow an Epstein-Barr viral (EBV) pharyngitis. Other diagnoses to be considered include peritonsillar or retropharyngeal abscesses and Ludwig angina. Lack of submental swelling, vocal changes,

airway compromise, or distant sites of infection point away from these conditions.

The treatment of Lemierre syndrome involves a 1- to 2-week course of IV antibiotics followed by 2 to 4 weeks of oral antibiotics, aimed at eradicating the endovascular infection. Initially use high doses of antimicrobial agents with good anaerobic coverage, such as clindamycin or metronidazole. *Fusobacterium* is also sensitive to penicillin and chloramphenicol. In one series, blood cultures were positive for MRSA. Therefore, initial treatment with vancomycin is justified. If medical management fails, surgical intervention to ligate the thrombosed vessel or drain abscesses may be needed. The use of heparin in Lemierre syndrome is debated, and no prospective studies are available. If jugular vein thrombosis has extended to the cavernous sinus, most experts agree that heparin is appropriate. Further treatment considerations are usually made in consultation with infectious disease and other specialty services.

Before antibiotics, Lemierre syndrome often led to a fatal septicemia within 2 weeks. Mortality from the syndrome is reportedly under 5%, but studies suggest that patients in whom the diagnosis was initially missed have a more ominous prognosis.

## KEY POINTS

- Think Lemierre syndrome when a patient in their late teens or early twenties presents with a recent or lingering sore throat and unilateral neck pain.
- The sore throat may be improving, but fever is usually still present.
- Pneumonia is the most common metastatic infection.
- Ten percent of cases follow EBV pharyngitis.
- ED ultrasound is a good screening test for jugular vein thrombophlebitis.

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## PERITONSILLAR ABSCESS

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**BEN LEESON, MD AND KIMBERLY LEESON, MD**

### INTRODUCTION

Peritonsillar abscess (PTA) is a common complication of acute tonsillitis. It occurs most often in adolescents and young adults but may occur in younger children. The estimated annual incidence is 30 per 100,000 persons aged 5 to 59 years of age, with 3 per 100,000 confirmed by aspiration of pus with drainage procedures. Approximately 45,000 cases occur each year in the United States.

PTA is an acute suppurative infection located between the palatine tonsils and the superior constrictor and the palatopharyngeus muscle of the pharynx. This condition should be suspected in patients who present with fever and severe sore throat. Diagnosis relies on clinical findings including trismus, uvular deviation, and inferior displacement of the superior pole of the tonsil on the affected side. A “hot potato” or muffled voice may be present as well as ipsilateral neck swelling and ear pain. Bilateral PTA is rare. Pediatric PTA diagnosis may be challenging since they can present atypically with more subtle symptoms.

### IMAGING

While imaging is not necessary to identify PTA, it may facilitate diagnosis and help delineate anatomy for treatment strategies. Consider imaging to distinguish cellulitis from abscess; evaluate for infection spread, in cases of inadequate exam due to severe trismus; and exclude other likely diagnoses.

- Computed tomography (CT) with IV contrast
- Lateral neck radiographs: assists when considering alternate diagnoses

- such as epiglottitis and retropharyngeal abscess
- Ultrasonography (helpful to distinguish PTA from cellulitis and guide needle aspiration)

## **MANAGEMENT**

### ***Evaluation Priorities***

The most important initial step in managing PTA is assessing the degree of upper airway obstruction. Patients who appear toxic, anxious, and with drooling and posturing must be monitored closely and may require prompt airway control. Consider a multidisciplinary approach to care for the sicker patients by involving ENT, surgery, and/or anesthesiology early in the ED course.

### ***Strategies***

- 1) PTA—aspiration/drainage, antibiotic therapy, and supportive care (hydration, analgesia, complication surveillance).
- 2) Probable PTA—attempt needle aspiration (preferred) or I&D and antibiotics.
- 3) Peritonsillar cellulitis—24-hour trial of antibiotics and supportive care with close follow-up.
- 4) Patients who fail to improve or deteriorate may require parenteral antibiotics and/or surgical intervention (tonsillectomy, I&D).

### ***Drainage Techniques (Aspiration, Incision and Drainage)***

PTA usually requires drainage through needle aspiration or less commonly I&D, which should be performed by an experienced physician. Needle aspiration is less painful and associated with less bleeding.

### ***Needle Aspiration***

Equipment needed: Cetacaine, lidocaine with epi, 5-mL syringe with 27-g needle, 18-g spinal needle, 10-mL control syringe, wall suction, Yankauer, tongue depressor, light source (can use disassembled plastic vaginal speculum with fiberoptic light or laryngoscope), emesis basin, cup of ice water, and straw.

- Ensure all equipment is available.
- Use tongue depressor or other device to ensure adequate visualization.
- Anesthetize the posterior pharynx with Cetacaine spray.
- Palpate and locate the area of maximal fluctuance.
- Inject area with 2 to 4 mL lidocaine with epi (27-g needle, 5-mL syringe)
- Cut cover on the 18-g spinal needle and replace, leaving 1.5 cm of needle exposed, and attach to a 10-mL control syringe.
- Aspirate the abscess, which is usually near the top of the tonsil lateral to the uvula.
- Have patient swish ice water to help reduce bleeding, and suction PRN.
- Send aspirate to the lab for culture and sensitivity.
- If bedside ultrasound is available, consider postprocedure imaging to confirm adequate drainage.

## ***Incision and Drainage***

May be considered using a scalpel as an alternate to needle aspiration. If this approach is used, wrap tape around the bottom portion of the 11 blade prior to use to minimize penetration and avoid vascular injury. Anesthetize and set up patient as for needle aspiration above. After the initial stab, use blunt dissection to express pus from abscess cavity.

## ***Antibiotic Therapy***

PTAs are often polymicrobial. Organisms include streptococcus (group A strep), *Staphylococcus aureus* (including MRSA), respiratory anaerobes, and occasionally *Haemophilus* species.

Oral (14 days)

- Amoxicillin-clavulanate
- Clindamycin

Parenteral

- Ampicillin-sulbactam
- Clindamycin
- Vancomycin (for suspected MRSA)

## ***Steroids***

Dexamethasone 10 mg plus IV clindamycin antibiotics resulted in less pain



at 24 hours compared to antibiotics alone. Also, return to normal activity and dietary intake was faster with steroids, but not statistically significant.

## ***Disposition***

- Outpatient management may be appropriate for older patients without complications who are well hydrated, can tolerate a drainage procedure, and tolerate oral intake including medications.
- Admit pediatric patients, those with impending airway compromise, complications, and comorbidities, and those who cannot tolerate oral intake.

## **COMPLICATIONS**

Complications, while rare, can occur with PTAs and can be life threatening. The key to avoiding complications is early diagnosis and appropriate treatment.

Complications of PTAs include:

- Airway obstruction
- Ludwig
- Sepsis
- Mediastinitis
- Aspiration PNA
- Internal jugular vein thrombosis
- IJ suppurative thrombophlebitis
- Sequelae of group A strep

Needle aspiration and I&D may also lead to complications, including carotid artery puncture/injury/bleeding. These concerns can be minimized by using a needle or scalpel guard that prevents the needle from puncturing deeper structures. Bedside ultrasound can be reassuring if direct visualization of the needle is maintained throughout the procedure. If ultrasound is not used during the procedure, one may perform an ultrasound after aspiration or I&D, to ensure that adequate drainage has occurred and that the abscess does not require further drainage. This is important since the recurrence rate of PTA is between 10% and 15%.

## **KEY POINTS**

- Be prepared for airway compromise.
- A patient-held laryngoscope or speculum light source after Cetacaine helps visualize and expose the posterior pharynx and frees the clinician's hands to perform aspiration or I&D more effectively.
- Use a spinal needle with needle guard to avoid vascular injury.
- Insert needle for aspiration more superiorly than expected.
- Ultrasound is useful to determine PTA size and location in relation to surrounding anatomy and can be used postprocedure to ensure adequate drainage.

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# DON'T MISDIAGNOSE, OVERTREAT, OR CAUSE PERFORATION OF THE TYMPANIC MEMBRANE

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JOHN HERRICK, DO

There are many causes of tympanic membrane (TM) perforation including otitis media, blunt trauma, barotrauma, and iatrogenic injury. While the treatment is often straightforward, there are some common mistakes to avoid.

The majority of perforations are caused by infection. Fluid and pressure build behind the TM causing pain. Eventually, the membrane ruptures, resulting in otorrhea. The presence of otorrhea in the absence of otitis externa confirms perforation, as the middle ear is normally air filled. The presence of pain also suggests infection and increased pressure behind the TM as simple perforations do not cause pain. Patients often feel a relief of pressure when the TM perforates in cases of otitis media.

Attempts at cleaning the ear are another common cause of TM perforation. Patients may insert cotton tip applicators, bobby pins, or other objects deep into the canal causing perforation. Health care workers can cause perforation with overzealous irrigation for cerumen impaction. Iatrogenic perforations are often large and are associated with delayed or incomplete healing.<sup>1</sup> Poor visualization while using instruments and patients who are unable to sit still may result in a perforation while attempting to remove a foreign body from the ear canal. For this reason, strongly consider procedural sedation prior to attempting removal. A button battery lodged in the ear can result in TM perforation. If not removed promptly, it can result in destruction of the ossicles and permanent hearing damage.<sup>2</sup>

A direct blow from an open hand, hitting the side of the head against

water, lightning strikes, blast injuries, and burns through the TM from stray welding slag are other well-documented causes of TM perforation. If the rupture is a result of scuba diving or water sports injury, there is an increased chance of infection due to contaminated water entering the middle ear and antibiotic prophylaxis is indicated. Do not clean the ear with soap as it lowers the surface tension and allows for easier penetration through the perforated TM.

Diagnosing TM perforation involves close inspection with an otoscope. A previous perforation may heal with a thin pseudomembrane. This may retract and be misdiagnosed as a persistent or new perforation. Air insufflation demonstrates decreased motion of the TM in the setting of perforation but may induce vertigo by pushing air into an injured otic capsule. Hearing should be tested. Rinne and Weber testing may help differentiate conductive and sensorineural hearing loss. Observe the patient for nystagmus, ataxia, and vertigo with head movement, which suggests deeper injury. Look for hemotympanum, bloody drainage, or leaking cerebrospinal fluid, which suggests basilar skull fracture.

CT with fine cuts of the temporal bones is the best test to evaluate for bony abnormality and fracture. CT with contrast can detect abscess or sigmoid sinus thrombosis, but MRI is the test of choice for infectious complications of the brain such as intracranial abscess or sinus thrombosis.

Treatment with systemic antibiotics for acute perforated OM should follow standard OM treatment guidelines with amoxicillin as the first-line agent.<sup>3</sup> Traumatic perforations that do not show signs of infection and have not been exposed to contaminated water do not require antibiotics. Topical therapy is a viable treatment for chronic suppurative otitis media (more common in adults than children).<sup>4</sup> Additionally, topical antibiotics are recommended for acute otitis media (AOM) in children with tympanostomy tubes. When topical agents are used, fluoroquinolones (ciprofloxacin, ofloxacin, or Ciprodex) are first line. Aminoglycosides such as neomycin/polymyxin B/hydrocortisone (Cortisporin Otic) are considered ototoxic and are not recommended as first-line agents. Neomycin can cause sensorineural hearing loss due to cochlear damage. The risk of ototoxicity is increased with prolonged use, so treatment should be limited to 10 days or less. Cortisporin Otic Suspension is recommended over the solution. The solution is more acidic and irritating, so it is contraindicated in a patient with a perforated TM. This product does not provide adequate coverage against the common pathogen *Streptococcus pneumoniae*. Patients should take precautions to keep water out of the ear.

Patients with new traumatic or large TM perforation should be referred to

an otolaryngologist for evaluation and audiometry if not available in ED. Most perforations will heal without treatment, but the size and location of the perforation help predict who may need repair. Indications for urgent consultation are ataxia, vertigo, significant hearing loss, and facial nerve impairment. Conditions warranting observation in the hospital include basilar skull fracture, ossicular disruption, facial paralysis, and perilymph fistula.

In the office, the otolaryngologist may patch the eardrum with paper and use a fat plug, fibrin glue, or Gelfoam. A tympanoplasty requires anesthesia and involves placing a graft in place of the injured TM. Surgery has a risk of further impairing hearing, so the risks and benefits must be assessed as many patients live with perforations without difficulty.

## KEY POINTS

- Most TM perforations are infectious or iatrogenic.
- Do not irrigate an ear with a known or suspected TM perforation.
- Clean, noninfected perforations do not require antibiotics.
- If antibiotics are required, systemic antibiotics should be used for AOM with perforation. Topical eardrops containing aminoglycosides and antibiotic solutions should be avoided.

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# IF IT AIN'T CANCER, WHY DO I CALL THIS MALIGNANT OTITIS EXTERNA?

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ALLISON D. LANE, MD

Malignant otitis externa (MOE), necrotizing otitis externa (NOE), and invasive otitis externa are all the same thing and not cancerous. So why is the common name of this condition malignant otitis externa? Well, in case you aren't aware, one of the actual definitions of the word "malignant" according to Merriam-Webster dictionary is "tending to produce death or deterioration." So while cancer is considered to be malignant, so is plague, or rabies, or life in general—none of us are getting out of here alive! But I digress. The condition was initially described by Meltzer and Kelemen in 1959 and later named and described by Chandler in 1968 as malignant external otitis due to its aggressive clinical behavior and high mortality rate approaching 50% at the time (now thought to be <10% for uncomplicated MOE). Some physicians have argued the name be changed from malignant to necrotizing or invasive, which would accurately describe the clinical nature of the disease but decrease the confusion of this being a cancerous condition.

MOE is an invasive infection of the external auditory canal and skull base, usually caused by *Pseudomonas aeruginosa*, with potentially life-threatening complications. MOE usually follows an external otitis, though it can also start with a middle ear infection. It is more common in males, age > 60 years, humid and warm climates, and nearly always with diabetes. Prognosis is worse for those with systemic immunodeficiencies.

Diabetes is the most significant risk factor for development of MOE. 90% of those with MOE diagnosis have diabetes. There is no difference in

predisposition between type I and II diabetics, nor is there necessarily a correlation to periods of hyperglycemia. Other forms of immunosuppression are also risk factors for the disease, for example, lymphoproliferative disorders and medications. And while patients with AIDS and MOE present with similar symptoms, these patients are generally younger, do not have diabetes, may not have granulation tissue in the canal, may have another dominant causative organism than *Pseudomonas*, and generally have a worse outcome.

Patients generally describe severe, deep-seated otalgia that may be worse at night, temporal headaches, and purulent otorrhea. Fever is uncommon. The most important findings on exam to suggest MOE are pain out of proportion to physical exam and granulation tissue at the floor of the osseocartilaginous junction. The latter is pathognomonic. The tympanic membrane is generally intact. Deterioration of mental status and cranial nerve abnormality may suggest intracranial complication.

Facial nerve, VII, is most commonly affected (about 20% incidence), though is not necessarily associated with a worse prognosis. Recovery from this palsy is, however, unpredictable and poor. With progression of the disease, nerves IX, X, XI, and XII may become affected, and later V and VI. Involvement of these additional cranial nerves carries an increased mortality rate.

Other intracranial complications are commonly fatal as they reflect severe disease. They rarely occur in the absence of cranial nerve palsies. Consider sigmoid sinus thrombosis, cavernous sinus thrombosis, meningitis, brain abscess, and dural sinus thrombosis.

Diagnosis of MOE is mostly clinical and may be supported by labs and imaging. The ESR will be elevated and can be used to support the clinical diagnosis, as simple otitis externa and ear canal malignancy usually do not cause elevation. CRP values are generally normal. CBC may show slight or no leukocytosis. Ear drainage culture and sensitivities should be performed before antimicrobial therapy if possible. 95% will show *P. aeruginosa*, though staphylococcal and fungal causes need to be considered.

CT imaging can prove extension of the infection to bone. Osteomyelitis may not be seen early on in the disease, since 30% to 50% of bone destruction is required for radiologic changes to become apparent. Suspicion must remain high even without bony changes. MRI may be useful for detecting suspected intracranial complications.

The mainstays of treatment for MOE include glucose control, correction of immunosuppression when possible, aural toilet, antimicrobial therapy, and in selected patients surgery. Consult with ENT early, if available. Admission

is likely required.

Otorrhea and debris can occlude the ear canal and should be cleared. Good methods include suction under direct visualization or fluffed cotton swab. A cotton wick can be placed to allow for drainage as well. Flushing is generally avoided as it can cause further irritation.

Patients with MOE will likely need admission for systemic antibiotics and never topical treatment alone. Topical treatment may disrupt normal flora, cause problems in isolating pathogens, and cause secondary fungal infections especially if combined with steroids. Empiric therapy should cover for *Pseudomonas* and *Staphylococcus*. High-dose therapy is needed due to poor vascularization of the target area, and a prolonged course is needed for osteomyelitis treatment. Most authors recommend treatment with oral ciprofloxacin 750 mg twice daily for 6 to 8 weeks, but a combination of agents may be needed. Surgery is reserved for local debridement, removal of bony sequestrum, or abscess drainage.

## KEY POINTS

- Typical bugs: *Pseudomonas* most common. Also consider *Staphylococcus* and fungus.
- Clinical diagnosis: granulation tissue at the floor of the osseocartilaginous junction and pain out of proportion to exam, usually in elderly diabetics.
- Workup: elevated ESR and CT with bony involvement.
- Complications: evaluate for signs and symptoms of intracranial extension.
- Treatment: prolonged course of systemic antimicrobials, not just topical.

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## APPROACH TO THE RED EYE

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LINDSEY RETTERATH, MD AND HANS BRADSHAW, MD

### I. Red Eyes that Require Immediate Ophthalmology Involvement

- A. Acute Angle-Closure Glaucoma
- B. Acute Anterior Uveitis
- C. Hyperacute Conjunctivitis

### ACUTE ANGLE-CLOSURE GLAUCOMA: WHEN TIME IS VISION, DON'T DELAY

This patient will likely be an older adult presenting in the evening (the hour of mydriasis) with the rapid onset of a painful red eye associated with seeing halos around lights, nausea, vomiting, and/or headache. Increased pressures can be felt on palpating the closed lid. Look for a nonreactive, *dilated pupil*. In hours, this red eye can progress to irreversible vision loss. Emergent ophthalmology involvement is paramount.

### ACUTE ANTERIOR UVEITIS: IMMEDIATE OPHTHALMOLOGY REFERRAL

This red eye presents as unilateral aching, photophobia, and blurry vision. Physical exam reveals redness where the iris meets the white of the eye and *pupillary constriction* with sluggish reactivity. On slit-lamp exam, note white cells and flare (fogging). Hypopyon develops as purulent debris settles in the anterior chamber. This can progress to glaucoma, pupil abnormalities, cataracts, macular dysfunction, and vision impairment. Start antibiotics and call ophthalmology quickly.

## **HYPERACUTE (GONOCOCCAL) CONJUNCTIVITIS: ANTIBIOTICS, IMMEDIATE REFERRAL**

This red eye progresses rapidly and involves copious exudate, lid swelling, and preauricular adenopathy. Patients often present when symptoms are unilateral. Treat topically (bacitracin, erythromycin, or ciprofloxacin) and systemically (1 shot ceftriaxone). Get ophthalmology involved to evaluate for corneal ulceration, which could lead to perforation.

Neonatal gonococcal conjunctivitis presents 2 to 5 days after delivery. Treat with one dose of ceftriaxone (cefotaxime if the baby has jaundice) and admit for close monitoring of potential corneal compromise.

### **II. Red Eyes that Warrant Reassurance, Noninvasive Treatment, or Outpatient Referral**

- A. Subconjunctival Hemorrhage
- B. (Nongonococcal) Conjunctivitis
- C. Blepharitis
- D. Episcleritis
- E. Scleritis
- F. Pterygium
- G. Superficial Keratitis

## **SUBCONJUNCTIVAL HEMORRHAGE: REASSURE**

When you see a unilateral, sharply demarcated red eye, ask about trauma, bleeding disorders, anticoagulation, retching, or hypertension. Pain may be present, but vision remains intact. Address underlying etiologies, but anticipate self-resolution of the hemorrhage. Symptoms beyond 3 weeks indicate ophthalmology follow-up.

## **CONJUNCTIVITIS: A COMMON PATHOLOGY WITH NUANCED ETIOLOGIES**

This diffusely red eye has nuanced causes and treatments. Notice dilated conjunctival vessels and discharge and +/- chemosis. Viral and bacterial conjunctivitis start unilaterally and then become bilateral. Clinical impression will direct management ([Table 138.1](#)).

**TABLE 138.1 VIRAL VS. BACTERIAL CONJUNCTIVITIS: OVERVIEW**

|                      | <b>VIRAL</b>                 | <b>BACTERIAL</b>  |
|----------------------|------------------------------|---|
| <b>Discharge</b>     | Clear                        | (Muco)purulent  |
| <b>Associated Sx</b> | Recent URI or viral prodrome | Lid and lash debris on waking (may also be present with adenovirus) |
| <b>Natural Hx</b>    | Self-resolving, 7–10 d       | Sx > 1 wk, improves w/ABx   |
| <b>Management</b>    | +/- prophylactic topical ABx | Therapeutic, topical ABx  |

## ***Viral Conjunctivitis***

**Viral conjunctivitis** is often managed with **prophylactic topical antibiotics** (trimethoprim and polymyxin B); however, return precautions suffice. Emphasize infection control: hand-washing, no shared towels, and no swimming for 2 weeks after onset. Persistence beyond 10 days merits an ophthalmology referral.

## ***Bacterial Conjunctivitis***

**Bacterial conjunctivitis** requires **topical antibiotics** (gentamicin or tobramycin). For severe infections, try ciprofloxacin or ofloxacin. One week of treatment without improvement merits an ophthalmology referral.

## ***Inclusion (Chlamydial) Conjunctivitis: Oral +/- topical antibiotics***

This mimics typical bacterial conjunctivitis. Suspect it in sexually active adults, especially with associated genitourinary symptoms or preauricular lymphadenopathy. The most specific finding would be follicles in the conjunctival fornix. Neonates will present 5 to 14 days after delivery with mucopurulent bloody discharge and/or pseudomembranes. Treat adults with oral tetracycline (avoid in pregnancy and peds), doxycycline, or erythromycin for 14 days (treat sexual partners, too!). In neonates, azithromycin for 3 days is an alternative.

## ***Allergic Conjunctivitis: Remove allergen, symptomatic relief***

These bilateral, pruritic red eyes associate with atopic disease, tearing, postnasal drip, and/or mucoid ocular discharge. An uncommon variation, “conjunctivitis medicamentosa,” involves a contact allergy to topical

medication and associates with swollen and scaling eyelids.

Topical levocabastine hydrochloride, systemic antihistamines, and artificial tears provide relief. Mast cell stabilizers have a slower onset but are preferred long term. Recommend allergen avoidance and PCP follow-up.

## **BLEPHARITIS: OUTPATIENT OPHTHALMOLOGY**

Blepharitis starts at the eyelash follicles and progresses to eyelid edema. This can cause ectropion or entropion (outward or inward lid deviation), tear instability, and irritated conjunctivae (red eye). Look for misdirected or fallen-out eyelashes. Usually chronic, this is best handled by outpatient ophthalmology.

## **EPISCLERITIS: REASSURANCE +/- NSAIDs**

Episcleritis presents as sudden-onset redness with tenderness to palpation. The sclera between inflamed episcleral vessels remains white. This is likely autoimmune and almost always self-limited. NSAIDs may help, but reassurance is sufficient. Persistent symptoms merit an ophthalmology referral.

## **SCLERITIS: PROMPT OUTPATIENT OPHTHALMOLOGY +/- NSAIDs**

This redness of the sclera itself associates with deeper pain than episcleritis. Like episcleritis, it is likely autoimmune; however, scleritis poses a threat to vision and requires prompt ophthalmology referral for likely systemic steroids or antimetabolites. Recommend NSAIDs for symptomatic management.

## **PTERYGIUM: ARTIFICIAL TEARS AND POSSIBLE REFERRAL**

This raised, fleshy, red-yellow, benign lesion results from conjunctival degeneration after prolonged sun and dust exposure. Artificial tears usually suffice, but outpatient ophthalmology is indicated for vision changes, acute enlargement, or suspected invasion into the cornea.

# SUPERFICIAL KERATITIS: LOOK FOR ETIOLOGY, OUTPATIENT OPHTHALMOLOGY

Dry eyes, medications, conjunctivitis, UV rays, contacts, and blepharitis can all cause superficial keratitis. Under fluorescein, look for uptake at multiple punctate lesions and/or a hazy cornea. Blurry vision and discomfort commonly associate. Cater management to the etiology (e.g., contact lens vacation). Refer to outpatient ophthalmology for definitive diagnosis and management.

## KEY POINTS

- Routinely ask about onset, progression, presence of pain, and vision changes.
- Routinely check visual acuity, pupils, and pressures.
- Call ophthalmology immediately if there's evidence of vision loss, severe pain, precipitous onset of symptoms, pupillary asymmetry, and findings suggestive of hypopyon.

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# EYEING THE CAUSES OF ACUTE VISION LOSS

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Acute vision loss is a frightening experience for our patients and a chief complaint that if not treated and triaged appropriately can lead to permanent vision loss. Etiologies may reside within the eye itself or represent manifestations of trauma, chemical exposure, or neurologic, cardiovascular, or inflammatory disease. Providers must cast a wide net and approach this complaint in a systematic fashion based on the patient's history. This chapter will focus primarily on etiologies within the eye.

A complete focused history for complaints of vision loss should include whether the vision loss is present in one or both eyes, whether it affects the entire field of vision, whether it is painful, speed of onset, and use of corrective lenses or contacts. Inquire regarding history of diabetes, neurologic or cardiac conditions, prior eye surgery or trauma, and any exposure to chemicals, irritants, or medications that can adversely affect the eye such as welding, power tools, or anticholinergic medications.

The physical exam should begin with assessing visual acuity in both eyes. If the patient usually wears corrective lenses, assess acuity using these lenses. If this is not possible, use a pinhole occluder instead. Report the smallest line for which the patient can read one half of the letters correctly, and how many letters were missed. If the patient suffered a chemical exposure, the first action should be extensive irrigation until effluent tests neutral on a pH strip. Evaluate extraocular movements, and ask if there is any pain with movement. Watch for asymmetric eye movement, specifically asymmetry causing double vision concerning for entrapment. When assessing pupillary response to light, perform a swinging flashlight test. Grossly approximate the patient's visual fields against your own. Apply

topical fluorescein to the eyes and inspect under a Wood lamp looking for increased dye uptake. Don't forget to evert the eyelids inspecting for lesions or foreign body. Intraocular pressure is measured directly on the cornea and not the sclera. Be sure to anesthetize the eyes first! Do not artificially skew your measurement by pressing down on the orbit or failing to calibrating your instrument.

The pathologies in this wide differential can be characterized by their presentation— whether the vision loss is painful or painless, present in one or both eyes. Remember to consider systemic causes as well as etiologies within the eye ([Table 139.1](#)).

**TABLE 139.1 DIFFERENTIAL DIAGNOSIS FOR VISION LOSS**

|                               | <b>PAINFUL</b>  | <b>PAINLESS</b>  |
|-------------------------------|---|--|
| <b>Unilateral Vision Loss</b> | <ul style="list-style-type: none"> <li>■ Corneal abrasion</li> <li>■ Acute angle-closure glaucoma<sup>a</sup></li> <li>■ Inflammation—iritis, uveitis</li> <li>■ Optic neuritis<sup>a</sup></li> <li>■ Endophthalmitis</li> <li>■ Cavernous sinus thrombosis</li> <li>■ Temporal arteritis<sup>a</sup></li> <li>■ Retro-orbital hematoma<sup>a</sup></li> <li>■ Infection—including zoster<sup>a</sup></li> <li>■ Toxic or caustic exposure to eye</li> <li>■ Trauma</li> </ul> | <ul style="list-style-type: none"> <li>■ Retinal detachment</li> <li>■ Vitreous detachment or hemorrhage</li> <li>■ Retinal artery or vein occlusion</li> <li>■ Lens dislocation</li> <li>■ Ischemic optic neuropathy</li> </ul> |
| <b>Bilateral Vision Loss</b>  | <ul style="list-style-type: none"> <li>■ Keratitis</li> <li>■ Optic chiasm impingement</li> <li>■ Toxic or caustic exposure to eye</li> <li>■ Trauma</li> </ul>   | <ul style="list-style-type: none"> <li>■ Stroke<sup>a</sup></li> <li>■ Medication side effects</li> <li>■ Metabolic derangement</li> <li>■ Keratitis</li> <li>■ Psychogenic</li> </ul>   |

<sup>a</sup> Indicates that this pathology is covered in depth in another chapter.

Acute glaucoma is a serious cause of painful unilateral vision loss that must be recognized quickly. The pain will often be described as a headache with a red, tearing eye. Remember the classic presentation of an older patient with pain that begins in a darkened room. Intraocular pressure will be significantly elevated.

Keratitis, a painful inflammation of the cornea, can be bacterial, viral, fungal, parasitic, or noninfectious. Look for new corneal opacities or ulcers



that should be visible without the slitlamp and take up fluorescein. Cell and flare may be present on slitlamp examination. Patients should immediately discontinue use of contact lenses. Same-day ophthalmologic referral is required for cultures and antibiotic selection.<sup>1</sup>

Uveitis is a painful inflammation of the iris, ciliary body, or choroid. Suspect uveitis when a ciliary flush of injection around the iris is present or when a bright line shining in the unaffected eye causes pain in the affected eye.<sup>1</sup> Slitlamp exam will reveal cell and flare.

Endophthalmitis refers to infection of the vitreous or aqueous humors, usually resulting from organisms introduced by trauma, surgery, or extension of keratitis.<sup>1</sup> Bacterial endophthalmitis most commonly presents within 1 week of cataract removal surgery<sup>2</sup> with white blood cells layering in the anterior chamber on slitlamp exam and a hazy view of the retina. Immediate ophthalmologic consultation and empiric antibiotics are required as this condition may result in severe visual impairment or even loss of the eye.<sup>2</sup>

Retinal detachment will often cause “floaters” to appear in the patient’s vision along with flashes of light in one or both eyes. This can be visualized under bedside ultrasound.<sup>3</sup>

Retinal artery occlusion usually results from carotid artery disease or embolism from atrial fibrillation and presents with sudden onset of profound painless vision loss in one eye over the course of seconds. A relative afferent pupillary defect or a cherry-red spot on a pale macula may be present.<sup>3</sup> Emergent referral should be sought.

Vitreous hemorrhage can occur in the setting of trauma, or spontaneously especially in the setting of sickle cell disease or pathologic neovascularization in diabetes.<sup>1</sup> Decreased red reflex may be present, red blood cells may be evident on slitlamp exam, and ultrasound may reveal hemorrhage in the posterior segment.<sup>1</sup> Check an INR in patients taking warfarin. Consult your ophthalmologist early.

## KEY POINTS

- Emergent referral is required for many potentially life-altering causes of vision loss. Early involvement of their expertise and equipment may save your patient’s eyesight.
- Always assess visual acuity in both eyes—it is the vital sign of the eye!

- Visual field deficits and afferent pupillary defects may reveal a patient's brain tumor—your physical exam matters.
- Do not ever send a patient home with topical ophthalmic anesthetics.
- Use bedside ultrasound when assessing for retinal detachment, retrobulbar hematoma, vitreous hemorrhage, and even extraocular movement if the eye is swollen shut.

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## FACE-EATING FUNGUS: RHINOCEREBRAL MUCORMYCOSIS

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**ERIC C. FUNK, MD AND CASEY M. CLEMENTS, MD,  
PhD**

Rhinocerebral mucormycosis is a rare but widely feared diagnosis, considering its high mortality and relatively poor treatment options. The most challenging aspect of the diagnosis of this disease is maintaining a high index of suspicion in susceptible patients. Additionally, the initial presentation of mucormycosis is often subtle and nonspecific.<sup>1</sup> The classic clinical presentation is an immunocompromised patient with a black eschar involving the nasal structures. However, the true presentation and predisposing conditions are often much more complicated. While mucormycosis can also cause highly morbid pulmonary or cutaneous disease, we will focus on the most feared: rhinocerebral infection.

The underlying theme in placing patients at risk for rhinocerebral mucormycosis is immune compromise.<sup>2</sup> The spores that give rise to this disease are ubiquitous in nature, but immunocompetent individuals are not affected despite frequent exposure.<sup>3</sup> Patients with diabetes, individuals having received stem cell or solid organ transplants, HIV/AIDS patients, and leukemia patients are at high risk. Severe malnutrition and IV drug abuse also put patients at higher risk. Victims of trauma are also more susceptible. In fact, there was a series of cutaneous mucormycosis in immunocompetent but traumatized victims of the Joplin, Missouri, tornado.<sup>4</sup> The fungal species that can cause mucormycosis rely on iron for growth, and therefore patients with iron overload are at high risk. Paradoxically, patients on deferoxamine are also at high risk, because the fungus has the ability to accept iron from the chelating agent.<sup>1</sup>

The initial presentation of mucormycosis may be difficult to identify. The classic presentation of a black eschar in the nose is helpful when present, but is found only rarely. Symptoms consistent with sinusitis are the most common finding. Additionally, the patient may report rhinorrhea, headache, midface pain, and facial edema. The challenge in diagnosing rhinocerebral mucormycosis is easy to understand when one considers the high number of diabetic patients presenting to the ED with nonspecific complaints. However, there are several key characteristics that help distinguish those patients that should raise the provider's suspicion. As the fungus spreads throughout the face and bones of the skull, vision changes and cranial nerve palsies may occur.<sup>3</sup> Neurologic changes are not common in most cases of sinusitis and should trigger a closer assessment. Findings consistent with periorbital cellulitis can occur as the fungus invades from the nasal mucosa into surrounding tissue. The worse the patients' diabetes control, the more likely they are to develop the infection. Patients with diabetic ketoacidosis and symptoms of sinusitis are at much higher risk than is a Type II diabetic with a mildly elevated hemoglobin A1C presenting with sinusitis. Susceptible patients who were previously evaluated in the ED for sinusitis and present with worsening symptoms should also be assessed more closely.

Workup for suspected mucormycosis includes labs and imaging, neither of which is definitive in ruling out infection. Screening labs are nonspecific, but may establish baselines that will be helpful during treatment. There are no laboratory blood tests available for diagnosing mucormycosis.<sup>1</sup> Imaging can be helpful in some cases. CT scan of the face will provide further assessment of sinuses and can show signs of bone infiltration or compromise. Nasopharyngeal scoping can identify intranasal involvement that is not visible externally.

Treatment options for rhinocerebral mucormycosis are limited, and many newer antifungals that we rely on are not effective. Urgent ENT consultation is warranted as surgical resection is almost always needed in addition to medical therapies. Amphotericin B remains the antifungal agent of choice. Posaconazole is being investigated, but has not been sufficiently studied to be recommended as first-line therapy.<sup>1</sup> Echinocandins, such as caspofungin and micafungin, have become commonly used in other fungal infections, but they are not likely to provide any benefit in mucormycosis. Atypical iron chelators, such as deferasirox, that do not make iron available to the offending fungus may be effective, but there is limited evidence for this approach. All patients with rhinocerebral mucormycosis require inpatient hospitalization for multidisciplinary management. Even so, outcomes remain very poor particularly in advanced disease. Rapid diagnosis gives patients the best chance.

A note about the nomenclature of mucormycosis: there has been a recent change in taxonomy of the infectious agents that cause mucormycosis. All offending fungal agents are now grouped into the subphyla mucormycotina.<sup>3</sup> While many clinicians may still use “mucor,” “rhizopus,” or “zygomycosis” to identify these diseases, it is important to note that these descriptors are not entirely accurate and may not appear in the genus or species names of the organism.

## KEY POINTS

- Maintain a high index of suspicion in immunocompromised patients.
- Initial symptoms will likely be subtle and may mimic sinusitis.
- Labs and imaging may not be diagnostic.
- Amphotericin B and surgical resection together are the cornerstones of therapy.
- Consult ENT early for management.

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## DIGGING FOR GOLD: SOME NUGGETS ABOUT EPISTAXIS

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**JOSH MUGELE, MD**

The first patient I encountered with epistaxis was on my first day of intern year. She was an elderly nun who was miserably holding a wad of tissues to her nose, trying to stop the bleeding, and I sat down with my clipboard to take a full H&P. Thankfully, my attending rushed in not long after and started shoving stuff up her nose. My lesson learned: when it comes to epistaxis, act fast.

Because epistaxis can be brisk and severe, it is important to have an algorithm you're comfortable with. It should include a stepwise approach and should include a combination of anesthesia, cautery, pressure, and vasoconstrictors. You should know what you have in your emergency department (ED), where it is located, and how to retrieve it quickly.

Most textbooks divide epistaxis into anterior and posterior. This breakdown isn't necessarily helpful to the emergency physician because anterior epistaxis may be copious and refractory to initial tamponade efforts, and similarly, posterior epistaxis may respond well to a large nasal tampon. It is more useful to think of epistaxis that can be controlled early in the ED and refractory epistaxis requiring more definitive intervention.

Because there are so many causes for epistaxis—URI, nose picking, minor trauma, dry air, CPAP, nasal steroids, cocaine, arteriovenous malformation, coagulopathies—the ED physician often has to MacGyver the response. Below is a discussion of a sample algorithm one might use in the ED.

### STEP 1

Have the patient pinch the nose at the alae and lean forward for 10 to 15 minutes while you gather supplies. When you return, have the patient gently blow his or her nose to remove any large clots. You might have to assist with forceps. If you can identify a small source of the bleeding—in many cases this will not be possible—anesthetize the nares with a topical anesthetic (deliver about 2 mL of lidocaine with epinephrine that you would use for suturing wounds via a nasal atomizer) and cauterize the site with silver nitrate sticks.

If the bleeding resolves with direct pressure or simple cautery, you may safely discharge the patient. Advise patients to similarly hold pressure at home and to time it on the clock (10 minutes can feel like a very long time). Depending on the history and reason for the bleed, you may recommend adjunct measures such as nasal saline spray, a short course of oxymetazoline, or a small amount of Vaseline or antibiotic ointment in each naris at bedtime.

## STEP 2

If bleeding is not controlled, move on to a combination of pressure and medication. Soak 2 to 3 cotton balls in a mixture of oxymetazoline and viscous lidocaine (or LET). Using the long ENT forceps, insert the cotton balls into the patient's affected naris (placed as far posteriorly as possible). Let the cotton balls sit in the naris for about 20 minutes, and then reassess as in step 1.

The combination of vasoconstrictor and pressure from the cotton balls is often sufficient to stop the bleeding given enough time. The lidocaine helps the patient tolerate the pressure and any additional packing or cautery that you may need to perform. You may use other vasoconstrictors such as epinephrine or cocaine depending on what you have available. Topical tranexamic acid is also an option.<sup>1</sup>

## STEP 3

If bleeding continues, pack the affected naris with a nasal tampon (or nasal balloon catheter). Common brands include Rapid Rhino, Rhino Rocket, and Merocel—these are equally effective.<sup>2,3</sup> Soak the tampon in sterile water, saline, or oxymetazoline to ease insertion. Avoid Vaseline or antibiotic ointment as this may counteract the prothrombotic surface on some products. Use the largest size available that will fit in the patient's naris, and aim posteriorly (not caudally) on insertion. Inflate the internal balloon with about 10 mL of saline or air.

Observe the patient for another half hour and check for bleeding around the tampon or in the back of the throat. If you observe continued bleeding, attempt to use a larger tampon or insert a second tampon in the opposite naris to provide additional tamponade. Patients with comorbidities, significant blood loss, or requiring bilateral packing should be admitted. Otherwise, you may safely discharge the patient home with ENT follow-up in 2 to 3 days. Antibiotics are usually not necessary (but may make your otolaryngologist happy).<sup>4</sup>

## STEP 4

If the epistaxis is unrelenting despite your best efforts, it's time for posterior tamponade. Most commercial posterior packing products use a double-balloon system—a large balloon for the anterior naris and a small balloon for the posterior naris. Many emergency physicians are familiar with using a Foley catheter for posterior bleeds by inserting the tip of the Foley into the nose until the balloon is in the oropharynx, inflating the balloon, and then pulling forward to lodge the balloon against the posterior bleed. An anterior tampon may then also be applied. This is extremely uncomfortable, so be generous with analgesia and anxiolysis (though beware of hemodynamic instability and vagal events).

Use caution when placing a device with a posterior balloon as it can fall back and obstruct the oropharynx, especially in patients with a high Mallampati class. Sit these patients upright and secure the posterior device with forward pressure using a hemostat that is taped to the face. You should obtain urgent ENT consultation, and these patients should be admitted to the ICU or step-down unit to observe for airway obstruction, hemodynamic stability, and vagal events. In all cases, if your patient is anticoagulated, make sure to draw coagulation labs and consider reversal for serious bleeding.

### KEY POINTS

- Have an algorithm and act fast.
- Be generous with topical and systemic analgesia.
- Use a combination of tamponade and vasoconstriction.
- Consider reversing patients on anticoagulation (or using a topical procoagulant).
- Always respect the posterior packing.



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## LUDWIG ANGINA—“THE GERMAN STRANGLEHOLD”

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DUSTIN LEIGH, MD

Ludwig angina is a rapidly progressive and frequently fatal gangrenous cellulitis of the soft tissues of the neck and floor of the mouth. First described in 1836, German physician Wilhelm Frederick von Ludwig considered this a “morbid entity.” While “Ludwig’s” is often loosely applied to deep space neck infections, it involves specific spaces and should be limited to those infections which are bilateral, involving the submandibular space (including both the sublingual and submylohyoid spaces). Prior to antibiotics, swelling frequently led to respiratory obstruction and death; thus, the term angina was added to the description, which arises from *angere* meaning “to strangle.” Identifiable source varies widely in the literature, ranging from 30% to 90% of cases having an identified source of infection. The submandibular space is the most common site, caused by odontogenic source in up to 85%. Other causes associated with development of deep space neck infection include laceration of the floor of the mouth, mandibular fracture, tumor, lymphadenitis, sialoadenitis, intravenous drugs injection, systemic infection, hematogenous spread of infection, or after foreign body ingestion.

An understanding of the anatomy of the cervical fascia is critical in identifying likely source and predicting extent and progression of infection. From a purely anatomic standpoint, these infections follow the path of least resistance, penetrating the nearest and thinnest tissue and tracking along the fascial planes in the neck and face. The deep cervical fascia in the neck is divided into superficial, middle, and deep layers. This tough connective tissue prevents the egress of pus toward the skin. As a result, infections will descend toward the mediastinum, ascend to the lateral pharynx and masticator spaces, or will expand to the point of airway obstruction.

Odynophagia is the most common presenting complaint (83.9% of patients). This is followed by dysphagia (71%), fever (67.7%), neck pain (54.8%), swelling (45.2%), trismus (38.7%), and respiratory distress (9.7%).

Physical examination can be organized into the following categories:

**General:** Overall level of comfort, note patient position; seated forward in the sniffing position is an ominous sign, and placing them supine may lead to complete airway collapse. Patients are often quite ill, some with associated shock states. Complete the physical with documentation of blood pressure, pulse, peripheral perfusion by assessment of capillary refill, skin temperature, and level of moisture.

**Mouth:** Visual assessment for symmetry, color, exudate of tonsils, posterior pharynx, uvula, and purulence of tonsils. Poor oral intake is the norm for these patients; comment on oral mucosa, moist or dry. Assess for sublingual edema, tongue elevation, and pooling of oral secretions. Closely inspect gums and teeth for gingival disease, dental caries, fracture, and purulent drainage with consideration for lower molars as frequent culprit. Trismus and limited interincisal opening should give you pause to attempt at orotracheal intubation as this is associated with very difficult airway.

**Neck:** Skin examination with visualization for swelling, erythema, ecchymosis, pustules, or “pointing” infection. Palpation of the anterior and posterior triangles should be performed. Range of motion may demonstrate hesitation with retropharyngeal involvement. Note appearance of unilateral JVD as jugular vein thrombosis is associated with infections in these spaces. Palpate the trachea to assess for position.

CT scanning is the most widely used modality for diagnosing these infections due to cost, availability, short acquisition time, and ability to localize abscesses in the head and neck, as well as other structural abnormalities.

*Streptococcus viridans* is the predominant organism in adult neck infection (43.7%) with *Klebsiella pneumonia* slightly more prevalent in diabetics (56.1%). Standard of care is to presume polymicrobial infection with empiric coverage for both aerobic and anaerobic infection. Empiric treatment regimens include clindamycin and beta lactamase alone or in combination with metronidazole. Of isolated species, nearly 20% are penicillin resistant and only 4% with resistance to clindamycin. Empiric antibiotic coverage must consider aerobic and anaerobic pathogens that synthesize beta lactamase. Second- or third-generation cephalosporin drugs

such as cefoxitin or ceftriaxone are effective.

Even in the modern antibiotic era, life-threatening complications, namely, airway compromise, jugular vein thrombosis, and descending mediastinitis, may develop due to delays in diagnosis and treatment. Predictors of complication include age > 65 (OR 6.12; 95% CI 1.63–22.89), diabetes mellitus (OR 9.0; 95% CI 2.08–38.95), other comorbidities (OR 5.44; 95% CI 1.72–17.17), and multiple space involvement (OR 10.80; 95% CI 2.59–44.97).

Airway compromise is the most immediate and life threatening of the complications encountered. Cases are described as “perilous airway.” Direct compression of the airway may arrive from displacement of the tongue posteriorly or secondary to laryngeal edema. These patients should be managed as presumed difficult airway and are challenging even when in a controlled OR environment. Up to 75% of these patients will need a tracheostomy. The decision to observe the airway, perform intubation, or perform tracheostomy must be made on an individual basis, considering the advantages and disadvantages of each.

## KEY POINTS

- Physical examination may be deceiving with few external clues of deep subfascial involvement.
- Early IV access with NPO and IVF.
- Infections polymicrobial; antibiotics to cover both aerobic and anaerobic.
- CT scan of the neck is the imaging modality of choice.
- Treat all patients as a difficult airway. Early involvement of ENT to assist with airway control and primary inpatient management.

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# **DENTAL EXAMS ARE NOT JUST FOR DENTISTS; REMEMBER TO IDENTIFY AND TREAT ORAL INFECTIONS**

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**ASHLEY SIEVERS, MD**

Oral exams are often daunting and overlooked. Is that tooth number 14 or the first molar? Which teeth are molars anyway? Regardless of your personal comfort with tooth numbering and naming, do not let this deter you from examining the oral cavity when patients present with dental pain or swelling.

The oral exam is not just for dentists. According to the American Dental Association, approximately one-third of Americans lack access to dental care. Unfortunately, the majority of these are people with chronic disease, the elderly, and the socioeconomically disadvantaged. We know that chronic diseases such as heart problems and diabetes are linked to poor dental health.

There are three stages of odontogenic infection including inoculation, cellulitis, and abscess. When a patient presents to the emergency department, it is often for evaluation of pain, swelling, or fever. They may present at any phase of infection, and an oral exam is the first step in distinguishing the infectious stage. Early in an infection, the pain is often described as a “toothache,” which becomes worse with temperature changes. With time, the pain may be described as more continuous and severe. An astute physician will consider the following: pain on tooth percussion, the presence or absence of trismus, fluctuant mass, fever, heart murmurs, and comorbid conditions.

## EXAM

Let's break the exam down further. Examine each tooth for tenderness to percussion and do not forget to consider the soft tissue spaces of the masticator and submandibular areas as well as the peritonsillar areas. Palpate the gingival mucosal areas on both the buccal (cheek) and lingual (tongue) side of each tooth for the presence of a fluctuant mass. Trismus is reduced opening of the jaw and is often caused by spasm, pain, or swelling. When trismus makes a detailed exam impossible, a CT scan can be utilized to identify causative teeth and the presence of abscess. One recent study using CT to identify abscess found that the most commonly involved cervical space infection was the masticator space, followed by the submandibular area.<sup>1</sup> Indications of more serious infection include fever, comorbid conditions (such as diabetes, advanced age, cardiovascular disease, and HIV), trismus, abnormal vital signs, and the presence of a heart murmur.

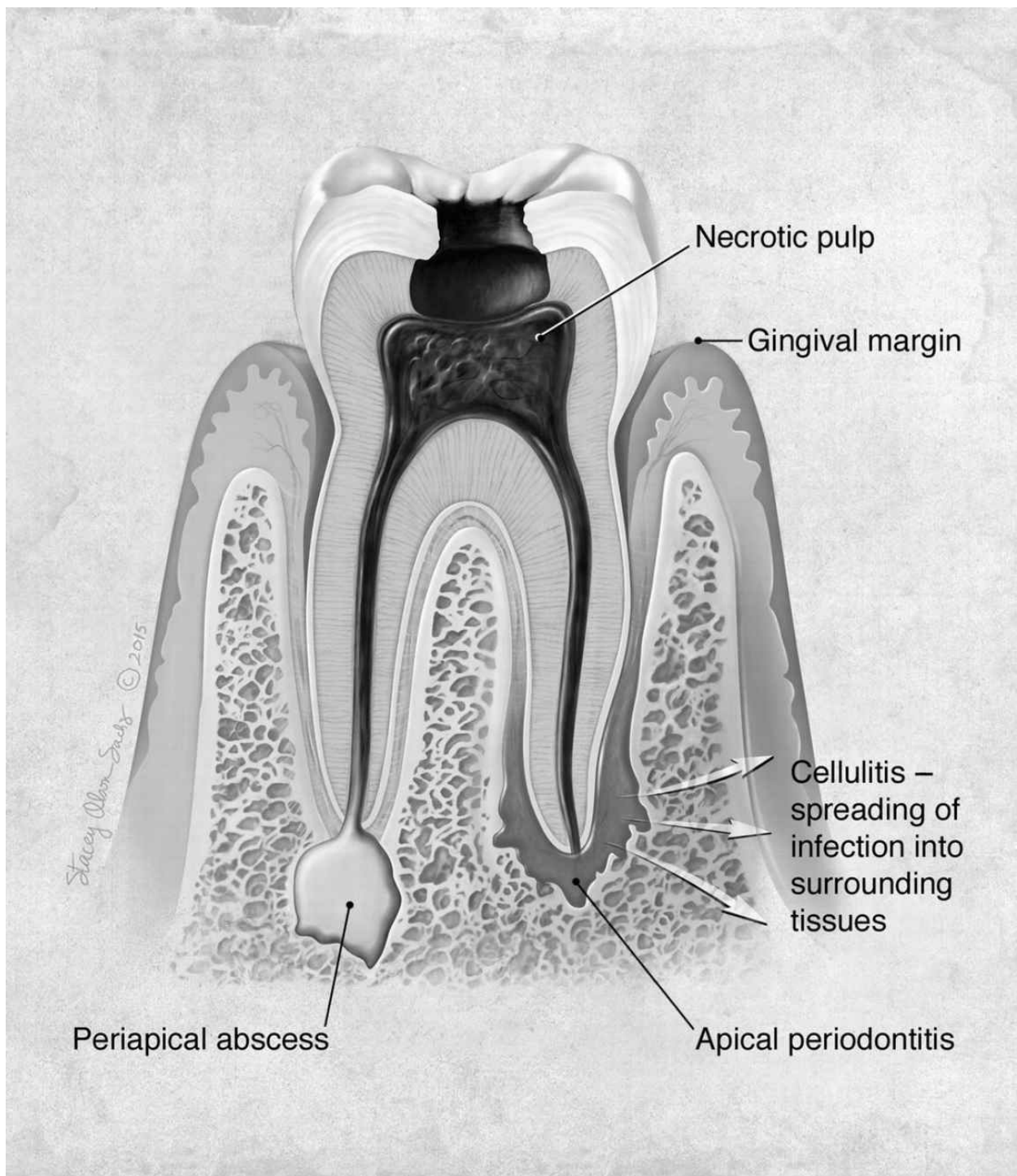
## IMAGING

Once an adequate exam has been performed, imaging can be considered. Panoramic x-ray, if available, is useful for identifying caries, but CT imaging will identify abscess formation. In a study of 4,209 patients with emergent odontogenic infections, 20.8% had abscess formation.<sup>2</sup> This number was substantially higher when trismus was present.

## ODONTOGENIC ABSCESS

Each odontogenic infection originates from plaque on the tooth surface. There are two areas of plaque entry, which results in two different types of infection.

Plaque may enter the tooth above the gingival margin (the area of the gums surrounding, but not attached to, the teeth) and lead to dental caries (cavities) that invade the deep tooth structure (pulp) and eventually disrupt the bone forming a periapical abscess ([Figure 143.1](#)).



**Figure 143.1**

Alternatively, plaque entering beneath the gingival margin may cause a periodontal abscess and is at risk of extension into the deep spaces of the neck. A periodontal abscess is different from a periapical because its source is the gums, not the tooth, and can be seen even in the absence of tooth decay. A significant and serious complication of both is their extension along planes of least resistance, which leads to deep space infections.

# TREATMENT

Once an infection is identified either by clinical exam or radiographic imaging, it must be properly treated. There are three components to treatment that yield satisfactory resolution of infection: empiric antibiotic therapy, surgical drainage of infection, and extraction or restoration of appropriate teeth involved. Though the emergency department is rarely able to provide tooth extraction, this is often a key component to definitive treatment. Any abscess identified does need to be drained as antibiotics alone will not sufficiently treat the infection. One recent study showed that abscesses in patients with multiple comorbidities are more likely to require multispecialty treatment.<sup>3</sup> Dental infections are usually polymicrobial with multiple studies showing the most frequent aerobic bacteria isolated are alpha hemolytic streptococci. Of the bacterial isolates, 69% are mixed aerobes and anaerobes with anaerobic streptococci followed by bacteroides being the most common anaerobic isolates. An acute abscess is usually preceded by acute apical periodontitis, and it is inferred that the microbial content would be of similar character. One study extracted 98 species from endodontic abscesses and showed 100% sensitivity to amoxicillin/ clavulanic acid and 91% susceptibility to amoxicillin alone (increased to 99% when combined with metronidazole). Clindamycin conferred 96% sensitivity.<sup>4</sup> It is vital to remember that microorganism susceptibilities vary from region to region, and this should be considered during antibiotic selection and targeted to the most likely organisms.

## KEY POINTS

- One-third of Americans lack access to proper dental care; a disproportionate number of these people are older and of poor socioeconomic status.
- Chronic poor oral health is associated with increased morbidity in coronary and cerebrovascular disease.
- Odontogenic infection may present to the ED anywhere along the spectrum of inoculation, cellulitis, or abscess. Remember to perform an oral exam.
- Proper treatment of an odontogenic infection is threefold and includes empiric antibiotics, drainage of abscess, and extraction (or surgical treatment of) of affected tooth. Most odontogenic infections are polymicrobial and susceptible to amoxicillin plus clavulanic acid or amoxicillin combined with metronidazole, but local sensitivities



should be considered in antibiotic selection.

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# THE INFECTION BEHIND THE INFECTION: DISTINGUISHING PERIORBITAL FROM ORBITAL CELLULITIS

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**SAMUEL J. TATE, MD AND JOHN S. ROSE, MD**

When a patient presents with redness to the skin around the eye, it can be a bit of a clinical conundrum. Is this a simple cellulitis of the skin or something more sinister that may extend into the deep orbital structures, or, worse yet, the brain? This is the challenge in distinguishing periorbital (or preseptal) from orbital (or postseptal) cellulitis. These two disease processes are divided anatomically by the orbital septum, which is a thin membrane of connective tissue that provides a barrier to potential infections from invading deeper structures: periorbital cellulitis anterior to and orbital cellulitis posterior to the septum.

Periorbital infections are made up of a constellation of infections of different anatomical structures. These include dacryocystitis, hordeolum, and cellulitis that is often initiated by mild skin trauma (like bug bites). These infections tend to be managed conservatively as an outpatient, but unique structures to the orbit can make these benign infections a setup for extension to badness. First, the valveless orbital veins allow for bidirectional flow potentially bringing remote infections to the orbit. Second, thin, permeable orbital walls predispose migration of local infection to deeper structures. Finally, proximity to nasal sinuses, particularly the ethmoid sinuses that are prone to complicated sinusitis, can be a reservoir for expanding infection.

Orbital cellulitis extends through the above routes and can cause orbital

abscess, subperiosteal abscess, osteomyelitis, cavernous sinus thrombosis, and even intracranial infections including meningitis, epidural or subdural abscess or empyema, and intracerebral abscess. These are obviously serious infections requiring admission, IV antibiotics, and potentially surgical intervention. The problem for the ED clinician is that periorbital and orbital cellulitis can present quite similarly, making these difficult to differentiate. Both are more common in children, but can affect adults as well. Both can present with periorbital edema and erythema plus or minus fever, URI, and rhinosinusitis and can be related to recent surgical procedures.

Luckily, unique presenting characteristics can differentiate these two. Preseptal infections tend to occur in younger patients (3.9 years) compared to orbital cellulitis (7.5 years). Acute sinusitis and fever were more common in orbital cellulitis (90% vs. 10% and 94% vs. 47%, respectively). In preseptal infections, the eye generally appears normal without any scleral injection or pain. A history of recent trauma, including insect bites, was more common in preseptal cellulitis (40% vs. 11%).<sup>1</sup>

Some “can’t miss” clinical signs should clue you into the presence of a deeper infection. Red flags such as diplopia, ophthalmoplegia, proptosis, and decreased visual acuity are extremely suggestive of orbital cellulitis. If present, these findings should prompt swift imaging and consultation to the appropriate service (ENT, ophthalmology, or neurosurgery).

Management of both preseptal and postseptal cellulitis is based on treatment of the underlying cause. *Haemophilus influenzae* type B was the overwhelming cause of both until widespread vaccination. H. flu should still be considered in unvaccinated patients and in children younger than 5 years of age as there is a much higher incidence in this population.<sup>2</sup> Currently, the most common source of infection is from typical strep, staph, and potentially MRSA.<sup>3</sup>

Management of periorbital cellulitis is first based on the EP’s clinical gestalt. If patient appears well and no red flags are present, then no imaging is indicated and treatment is directed at the underlying cause. For example, if patient has dacryocystitis, you should obtain Gram stain and cultures of the fluid and start empiric antibiotics to cover strep and staph.

If there is suspicion for orbital cellulitis, a contrast-enhanced CT of orbits and sinuses should be quickly obtained. If the patient is sick, promptly cover with a third-generation cephalosporin and vancomycin or clindamycin empirically. Blood cultures have low yield, and typically fluid cultures or intraoperative fluid cultures will guide management for the inpatient team although this may be institution specific. Consultation and admission should

be obtained based on the imaging results.

As orbital cellulitis can be a very complicated infection, there are several “can’t miss” complications or potential mimics that should be considered on all of these patients. First, cavernous sinus thrombosis (CST) presents very similarly with proptosis, ophthalmoplegia, and loss of vision. CST also may present with cranial nerve palsy of III, IV, V, and VI with CN VI being most common, but it has a notoriously variable presentation. MRI/MRV of the brain is the test of choice, while treatment with similar antibiotics to orbital cellulitis should be started. Heparin is controversial, so decision should be made in conjunction with a specialist. Second, orbital pseudotumor may present similarly, but is really an idiopathic inflammation and requires corticosteroids for treatment. Third, herpes zoster ophthalmicus may present with erythema and vision changes and would require antiviral medications.

The most concerning complication of periorbital cellulitis is recurrent episodes known as RPOC defined as three infections in a year with at least 1 month between episodes. These presentations should prompt a search for atypical causes including HSV, fungal, HIV, mycobacteria, and neoplasm.

Orbital and periorbital cellulitis can be difficult to differentiate, but trust your clinical acumen, let your imaging direct your management (if needed), and remember to always consider red flags and rule out the “can’t miss” complications.

## KEY POINTS

- Periorbital cellulitis treatment is targeted at the underlying cause and does not need imaging.
- Red flags for orbital cellulitis include diplopia, ophthalmoplegia, proptosis, or decreased visual acuity.
- Contrast-enhanced CT of orbits and sinuses is imaging of choice for concern for orbital cellulitis.
- Most common causes of both preseptal and postseptal cellulitis are strep and staph, but consider H. flu in unvaccinated and <5-year-old children.
- Be suspicious of CST and consider MRI/MRV of brain.

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# **SECTION XI**

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# **HEME ONC**

# **WHEN KIDNEYS EXPLODE; EVERYTHING IS WRONG WITH TUMOR LYSIS SYNDROME**

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**DANIEL CABRERA, MD**

## **WHAT IS TLS?**

Tumor lysis syndrome (TLS) is a metabolic syndrome characterized by renal failure and multiple metabolic abnormalities caused by the rapid and massive cell destruction and release of intracellular content into the plasma. It typically occurs on rapidly aggressive hematologic malignancies or after treatment initiation in some solid tumors, such as high-grade lymphomas. TLS is probably one of the most common oncologic emergencies, with a spectrum of presentations ranging from minimally symptomatic to life threatening; its rapid recognition and treatment are essential for Emergency Physicians.

The pathogenesis of TLS rests in the immense cell lysis after the initiation of therapy, commonly chemotherapy, although radiotherapy and steroids occasionally precipitate it as well. The syndrome is a combination of the electrolytic and inflammatory effects of the release of intracellular contents (potassium, phosphorus, and nucleic acids) as well as renal failure that follows secondary to crystal precipitation in the renal tubules, inflammatory mediator-driven vasoconstriction, and concomitant hypovolemia.

## **OPERATIONAL DEFINITION**

A universal definition of TLS has been difficult to achieve, and some controversy exists about the core characteristics of the syndrome. Currently, the Cairo and Bishop classification ([Table 145.1](#)) is accepted as the standard norm and distinguishes between laboratory and clinical TLS. In the regular emergency medicine practice, the distinction is not particularly helpful, but it is important to know it in order to have a cogent discussion with your nephrology or oncology consultant and to identify patients at risk of the disease who may benefit from close follow-up.

**TABLE 145.1 CAIRO-BISHOP CLASSIFICATION OF TUMOR LYSIS SYNDROME**

| LABORATORY TLS              | CLINICAL TLS  |
|-----------------------------|---|
| Uric acid $\geq 8.0$ mg/dL  | AKI (defined as creatinine $> 1.5\times$ the upper limit of normal for patient age and sex) |
| Potassium $\geq 6.0$ mEq/dL | Cardiac arrhythmia  |
| Phosphorus $\geq 4.6$ mg/dL | Seizure, tetany, or other symptomatic hypocalcemia  |
| Calcium $\leq 7.0$ mg/dL    |   |

## WHEN TO SUSPECT AND HOW TO DIAGNOSE?

The symptoms of TLS are often nonspecific and when recognizable are related to hyperkalemia, hypovolemia, hypocalcemia, and renal failure. This syndrome is commonly underdiagnosed, and the major challenge is its identification; the diagnosis should be at least entertained in any patient with recent initiation of therapy for malignancy. A common mistake is to diagnose acute renal failure in these patients and move to *treatment mode* without looking into the cause.

Recognizing TLS is important as there is specific therapy that can make a difference. In others words, we need to overcome satisfaction bias and consider TLS as the etiology of new acute renal failure in oncologic patients. It is probably advisable to obtain a serum uric acid in all sick-appearing oncologic patients with new-onset renal failure.

## AVOID KIDNEY EXPLOSION

Although outside of the scope of control of emergency physicians, the prevention of TLS with pretreatment hydration and allopurinol is of paramount importance and a key detail in the clinical history to obtain.



Patients who have mild clinical symptoms, are well appearing, and have normal or minimal renal dysfunction but developed chemical abnormalities should probably be admitted for hydration, allopurinol, and electrolyte monitoring. Patients with a similar clinical profile but with more laboratory abnormalities and signs of renal dysfunction should receive aggressive fluids, rasburicase, admission to an ICU, and consultation with oncology and nephrology.

The pharmacologic treatment of TLS is based on allopurinol and rasburicase. Allopurinol is a xanthine oxidase inhibitor that prevents the conversion of xanthines into uric acid; therefore it prevents the new uric acid formation but does not alter the acid already in the plasma. Rasburicase is a recombinant urate oxidase that converts uric acid into allantoin, making it more soluble and facilitating the excretion 5 to 10 times; the major issue is that the drug is very expensive and in some centers requires approval by oncology or nephrology before its use.

## KEEP THINGS SIMPLE

The main problem is the patient with TLS who is clinically *sick*. Typically, these patients are extremely fragile, and management needs to be extremely precise, but at the same time, the overwhelming metabolic derangements can be, well, overwhelming.

The best way to approach this is to proceed into a step-by-step approach (see also [Table 145.2](#)):

**TABLE 145.2 TREATMENT OF METABOLIC ABNORMALITIES ASSOCIATED WITH TLS**

|                   |                    |   |
|-------------------|--------------------|---|
| Hypovolemia       | Intravenous fluids | Aggressive until urinary output of 1 mL/kg/h IV is achieved |
| Renal failure     | Dialysis           |   |
| Hyperuricemia     | Allopurinol        | 600 mg initially followed by 300 mg/daily IV                |
|                   | Rasburicase        | 0.05–0.2 mg/kg IV   |
| Hyperphosphatemia | Aluminum hydroxide | 100 mg/kg/d PO  |
| Hyperkalemia      | Standard treatment |   |
| Hypocalcemia      | Calcium gluconate  | 50–200 mg IV  |

Adapted from Halfdanarson TR, Hogan WJ, Moynihan TJ. Oncologic emergencies: diagnosis and treatment (Review). *Mayo Clin Proc.* 2006;81(6):835–848.

- Take a deep breath and keep it simple stupid.

- Contact nephrology as soon as possible as this patient may require hemodialysis soon.
- Start working on an ICU bed.
- Correct the hyperkalemia as soon as possible using your standard *catastrophic* hyperkalemia approach, but consider hemodialysis early.
- Correct hypovolemia in a *septic shock–like aggressive fashion*. Recommendations are 30 to 40 mL/kg until around 100 mL/h of urine output is achieved.
- Start allopurinol and ideally also use rasburicase as it will inhibit the production of uric acid and will help to excrete already formed urates.
- Attempt to correct the hypocalcemia; however, this could be challenging and may require hemodialysis.

Hemodialysis in patients with TLS is frequent and usually is recommended more liberally compared to other renal emergencies; involving your *emergency nephrologist* early in the game is advisable. Among the indications of renal replacement are oligoanuria, volume overload, refractory hypocalcemia, and severe hyperkalemia.

## KEY POINTS

- TLS is more common than you may think in oncologic patients.
- Consider TLS in oncologic patients who are sick or with new renal failure.
- Aggressive fluids, allopurinol, and rasburicase are the main treatment.
- Don't get overwhelmed by the magnitude of derangements; take care of the problems in order of lethality.
- Consider hemodialysis early.

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# IMMUNE THROMBOCYTOPENIA: OH THE PLATELETS, YOU'LL Go!

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NICOLE MUHLBAUER, MD, MPH AND NEHA BHASIN, MD

When a patient presents to your emergency department with a platelet count of  $15 \times 10^9/\text{L}$ , you will likely have a strong urge to fix the platelet count as soon as possible, or admit the patient so that the hematologist can take over. However, this may not be necessary.

The diagnosis of immune thrombocytopenia (ITP) may be made by history, physical exam, and a platelet count. ITP is characterized by a platelet count  $<100 \times 10^9/\text{L}$ . It is categorized as either primary or secondary. Primary ITP, the more common type in the pediatric population, is the presence of thrombocytopenia in an otherwise healthy individual without an underlying cause. With thorough history taking, a recent viral illness is typically uncovered. The pediatric population with ITP typically presents at  $<10$  years of age. Secondary ITP, more common in adolescents and adults, can occur as a presenting sign of an underlying autoimmune disease, malignancy, acute infection or drug reaction.

Although the exact mechanisms have yet to be made clear, one fact is obvious. Platelets are being destroyed at a faster rate than they are being produced. Clinically, hemostasis is disrupted.

The development of a petechial rash or mucosal bleeding will prompt the ITP patient to present to the emergency department. Unless the patient is actively bleeding, a patient with ITP is well appearing and hemodynamically stable. This is important because it helps the physician differentiate from other diagnoses that also have a petechial rash, such as an infectious cause like meningococcemia. Similarly, oncologic processes are in the differential

as well. An ITP patient lacks lymphadenopathy, weight loss, or night sweats. Furthermore, a complete blood count reveals an isolated thrombocytopenia with preservation of the remaining cell lines.

In the absence of red flags for infection or malignancy, additional lab work is not required for the pediatric population with ITP. However, in the adult patients, an underlying infectious or malignant pathology is common. Therefore, HIV and HCV testing is recommended for the adult patient. The American Academy of Hematology does not recommend a bone marrow examination or platelet antibody testing for the diagnosis to be made in either population.

Once the diagnosis of ITP is made, observation should be the mainstay management for the adult and pediatric patient who is asymptomatic or only has bruising or petechiae of the skin, regardless of the platelet count. The majority of pediatric patients will have a spontaneous remission within 6 months. Unfortunately, platelet counts are less likely to recover spontaneously in adolescents and adults with ITP. Treatment may be initiated for adult patients with a platelet count of  $<30 \times 10^9/L$ .

The most dreaded complication of ITP is an intracranial hemorrhage. When it occurs, it results in significant neurologic impairment or death. It is typically an intraparenchymal bleed. Less than 1% of all the patients who have ITP will develop an intracranial bleed, and the majority of occurrences take place in patients with a platelet count  $<10 \times 10^9/L$ . Although the majority of intracranial bleeds occur with very low platelets, one cannot rely on the number of platelets because it is primarily platelet function that will dictate hemostasis for the patient. Thus, the tendency to have a severe bleed is varied among patients and not directly related to the platelet count. Currently, there does not exist a risk assessment tool for practitioners to utilize when a patient presents to the emergency department. Therefore, many practitioners will choose to treat thrombocytopenia  $<10 \times 10^9/L$ .

The commonly chosen treatment options for a hemodynamically stable patient include intravenous immunoglobulin (IVIG) and corticosteroids. IVIG has been shown to increase the platelet count faster compared to steroids. The recommended IVIG dose is 0.8 to 1.0 g/kg. If treating with IVIG, you may consider premedication with diphenhydramine and acetaminophen for side effects such as headache and nausea. If headache persists despite these interventions and slowing the infusion rate, consider ordering an emergent noncontrast CT of the brain to evaluate for an intracranial hemorrhage. There is significant variability among doses for corticosteroids. Many practitioners will use a dose of 1 to 2 mg/kg/d for a period of up to 4 weeks with a taper. Patients must be informed that the

platelets may or may not recover quickly and activities with increased risk of bleeding should be avoided.

For all patients presenting to the emergency department with an acute bleed, ABCs remain paramount. When you discover that the patient is also thrombocytopenic, it is recommended to immediately transfuse platelets rapidly and repeatedly as needed. This may be completed simultaneously with an infusion IVIG, if ITP is suspected. Additional interventions presented in case reports include recombinant factor VIIa and antifibrinolytic agents, or emergent splenectomy may serve as a last ditch effort to save the patient. Clearly, each intervention carries its own risks.

## KEY POINTS

- Primary ITP is more common in otherwise healthy children and may be preceded by viral illness.
- Secondary ITP is more common in adolescents and adults and may be associated with infection, malignancy, or autoimmune disease. Test to rule out HIV and HCV.
- Limited workup and observation are the reasonable first-line approach in the pediatric patient, but not for the adult because of the increased association with malignancy or infection.
- There is no risk assessment available to assess a patient's risk of intracranial hemorrhage.
- IVIG and corticosteroids remain the mainstay of treatment.

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# **THROMBOTIC THROMBOCYTOPENIC PURPURA AND HEMOLYTIC UREMIC SYNDROME: BLOODY ZEBRAS WITH A BAD BITE**

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**STEPHEN A. MANGANARO, FACEP, FAAFP**

Thrombotic thrombocytopenic purpura (TTP) and hemolytic uremic syndrome (HUS) are rarely seen platelet aggregation disorders involving the deposition of fibrin and tiny clots in capillaries and arterioles leading to microangiopathic hemolytic anemia through the shearing of RBCs and to a systemic depletion of platelets, accompanied by end organ damage due to reduced circulation.

Although TTP and HUS are similar disorders, TTP is more common in adults and has more prominent neurologic effects. HUS is more often seen in children, and renal effects predominate.

## **THROMBOTIC THROMBOCYTOPENIC PURPURA**

The cause of TTP is not well understood. For reasons yet unknown, ADAMTS13 (an enzyme that normally breaks down von Willebrand factor) is inhibited, which leads to the formation of microemboli. About 40% of cases appearing to be secondary to other conditions, including cancer, pregnancy, autoimmune diseases, HIV, influenza vaccinations, and certain medications (quinine, acyclovir, clopidogrel, and certain

immunosuppressants). It may also have an association with bone marrow transplants. In general, there is a 60% female predominance. There also appears to be a hereditary form of the disorder.

Along with being something we don't see often, TTP can have a very diverse presentation and range from mild to severe. The "Classic Pentad" of TTP is as follows:

- 1) Fever is seen in ~90% of cases.
- 2) Thrombocytopenia (10,000 to 50,000), with purpura.
- 3) Microangiopathic hemolytic anemia with evidence of fragmentation of RBCs (schistocytes) on the blood smear.
- 4) Fluctuating neurologic signs that may include stroke, seizures, altered mental status, and coma (one-third of patients may not have neurologic signs).
- 5) Renal compromise—but often the creatinine remains normal or shows only an insignificant, transient rise.

The anemia seen with TTP will usually manifest with a hematocrit below 20, and schistocytes (fragmented red cells) can often be detected on smears. The degree of renal involvement in TTP can vary from hematuria and proteinuria to frank acute renal failure.

The treatment of TTP with plasma exchange using fresh frozen plasma has markedly improved the prognosis of this condition. In addition to plasma exchange, initial therapy may include steroids and antiplatelet agents such as aspirin. Unless there is life-threatening hemorrhage present, platelet transfusions should be avoided because the same platelet destructive process will affect the transfused platelets and cause additional thrombi to form in the microcirculation.

Before the use of plasma replacement infusion became widespread, mortality from TTP reached as high as 90%. With modern treatment, the mortality from this condition has fallen below 20%. Relapse is not uncommon and has been observed to occur in 20% to 50% of patients >30 days following remission. Emergency physicians must keep a sharp eye out for the return of signs and symptoms in patients with a prior history of TTP.

## HEMOLYTIC UREMIC SYNDROME

HUS is one of the most common causes of acute renal failure in childhood. The clinical triad consists of a microangiopathic hemolytic anemia, renal failure, and thrombocytopenia. A typical presentation includes watery diarrhea, crampy abdominal pain, and possible fever. Five to ten days after



onset of symptoms, patients often experience increased abdominal pain, blood stools (colitis), hemolytic anemia, thrombocytopenia, and acute renal insufficiency with possible progression to renal failure.

The most common causative agent of HUS is a shigatoxin-producing *Escherichia coli* (STEC). In the US, the culprit is often *E. coli* serotype 0157:H7 found in frozen ground beef patties, frozen pepperoni pizza, bagged fresh spinach, as well as lettuce, cheddar, and ground beef in fast food. Unlike TTP, the mechanism is simpler: the shigatoxin is known to cause endothelial damage, leukocyte activation, platelet activation, and widespread inflammation. These processes all promote the formation of thrombi in small vessels that lead to microangiopathic hemolytic anemia and thrombocytopenia. Other causes of HUS include primary disorders of complement regulation (referred to as atypical HUS), other infections including *Streptococcus pneumoniae* and HIV, as well as drug toxicity particularly in patients with cancer or solid organ transplant recipients.

The treatment of HUS is primarily supportive and includes early intravenous fluids for rehydration (with a caution to avoid overhydration). Platelet or RBC transfusion is recommended only for severe active bleeding situations. Should renal failure develop, hemodialysis or peritoneal dialysis may be required. Antimotility drugs should be avoided as these may lead to toxic megacolon. Antibiotics may increase the release of shigatoxin from the bacteria and should be avoided. Though neurologic symptoms are less prominent than in TTP, they can be seen in up to 25% of HUS patients.

The good news is that early dialysis and supportive therapy result in return to baseline renal function in 90% of patients. Unlike TTP, recurrence of (typical) HUS is uncommon.

Diagnosis of these two conditions is based on the spectrum of clinical symptoms as well as certain diagnostic tests. Since TTP is associated with ADAMTS13 inhibition, a lab test showing <5% of normal ADAMTS13 levels is indicative of TTP. ADAMTS13 levels >5% plus shigatoxin-positive stool studies point toward typical HUS, while shiga-negative patients with levels >5% may have atypical HUS.

Here follows a table that compares and contrasts these two “zebroid” but crucial-to-recognize conditions ([Table 147.1](#)).

|   |
|---|
| <b>TABLE 147.1 COMPARISON HUS vs. TTP</b> |
|---|

|                  | <b>HUS</b>                 | <b>TTP</b>                 |
|------------------|----------------------------|----------------------------|
| Age              | Children                   | Adults                     |
| CBC              | Anemia<br>Thrombocytopenia | Anemia<br>Thrombocytopenia |
| Peripheral Smear | MAHA<br>Schistocytes       | MAHA<br>Schistocytes       |
| Clinical         | Predominantly renal        | Predominantly CNS          |
| Treatment        | Supportive<br>Dialysis     | Plasmapheresis<br>Steroids |
| Prognosis        | Better                     | Worse                      |

## KEY POINTS

- TTP and HUS are both platelet aggregation disorders.
- TTP is more common in adults and has more prominent neurologic effects.
- HUS is a common cause of renal failure in children.
- The most common causative agent of HUS is a shigatoxin-producing *E. coli*.
- HUS is treated supportively (with dialysis as needed) and has the better prognosis.
- Think TTP or HUS when confronted with anemia, thrombocytopenia, and schistocytes.

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# **HIGH TEMPS AND LOW COUNTS: TREAT FEBRILE NEUTROPENIC PATIENTS WITH EARLY AND APPROPRIATE ANTIBIOTICS**

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**MATTHEW W. CONNELLY, MD AND STEVEN ROUMPF, MD**

When faced with neutropenic fever, the gut reaction of many emergency medicine physicians is to wage chemical warfare on any and all possible infectious causes. As nearly half of these patients will have an identifiable source of infection, efficient evaluation and recognition are key to early initiation of treatment. The clinical challenge in treating patients with neutropenic fever lies in carefully managing the balance between early, sufficiently broad antibiotic coverage and the ever-increasing rates of antibiotic-resistant organisms. The 2010 update from the Infectious Disease Society of America guidelines continues to promote early empiric antibiotic treatment in the febrile neutropenic patient; however, more emphasis is now being placed on selecting proper antimicrobial therapy on a case-by-case basis rather than a “shotgun” approach. A recent move toward stratifying febrile neutropenic patients into high- and low-risk categories, based on various presenting factors and clinical gestalt, may help to direct physicians to the appropriate empiric treatment agents and disposition.

Neutropenic fever is most commonly defined as an absolute neutrophil count (ANC) of  $<500$  cells/mm<sup>3</sup> or an ANC that is expected to decrease below 500 cells/mm<sup>3</sup> during the next 48 hours and a single oral temperature measurement of  $>38.3^{\circ}\text{C}$  ( $101^{\circ}\text{F}$ ) or a temperature of  $>38.0^{\circ}\text{C}$  ( $100.4^{\circ}\text{F}$ )

sustained over a 60-minute period. Neutropenia is further classified as profound when the ANC falls below 100 cells/mm<sup>3</sup>. Providers must also be able to recognize signs of infection in the afebrile neutropenic patients and treat them similarly to those with documented fever.<sup>1</sup> Once patients meeting these definitions have been identified, chest x-ray and pan-cultures, including blood from peripheral and central lines (all ports), urine, sputum, and CSF if there is concern for meningitis, should be obtained and individuals can be stratified into low- and high-risks groups based on multiple factors.

The Multinational Association of Supportive Care in Cancer risk-index score has been established and validated to quantify the likelihood of serious complications using objective criteria as well as subjective patient clinical status as determined by the provider.<sup>2</sup> Low-risk characteristics in the otherwise healthy, well-appearing patient include age < 60, outpatient status, solid tumors, or hematologic malignancy with no history of fungal infection. Further criteria include ability to tolerate oral medications and expected improvement of ANC within the next 7 days. This can be predicted, in consultation with oncology, by considering ANC response to previous chemotherapy, timing of future treatment, and receipt of bone marrow stimulating medications. High-risk patients are characterized by age > 60, history of COPD, acute gastrointestinal and/or neurologic symptoms, and signs of dehydration or hypotension. Profound neutropenia expected to last longer than 7 days and increased duration of neutropenia have also been found to have high rates of serious infection.<sup>1</sup> Once stratified and stable, options regarding treatment and disposition can be discussed with the patient and his or her oncologist.

Low-risk patients with the proper home support structure and close follow-up (within 24 hours) may be discharged home on oral ciprofloxacin plus amoxicillin/clavulanic acid after discussion with their oncologist. Clindamycin may be substituted for amoxicillin/clavulanic acid in penicillin allergic patients. Early differentiation of the cliché “sick vs. not sick” may help to save these patients the cost and side effects associated with IV antibiotic therapy. For patients deemed to be at high risk for significant infection, no literature currently exists regarding a single superior empiric IV antibiotic regimen; however, numerous monotherapies have shown a high level of efficacy despite increasing incidence of antibiotic-resistant organisms. An expanding body of evidence has supported the use of antipseudomonal  $\beta$ -lactam agents such as piperacillin-tazobactam, carbapenems (meropenem or imipenem-cilastin), or cefepime, keeping in mind institutional pathogen susceptibility. For patients with a history of severe penicillin allergy, empiric treatment with ciprofloxacin PLUS

clindamycin or aztreonam PLUS vancomycin is recommended. As coagulase-negative staphylococci are the identifiable causative agent in the majority of bacteremia in neutropenic patients, and will be covered by  $\beta$ -lactams, vancomycin should not be part of standard empiric therapy and has not been shown to significantly alter mortality rates in this population. Addition of vancomycin (or linezolid/daptomycin in cases of known VRE) should be considered in cases of hemodynamic instability, radiographic pneumonia, suspected vascular catheter or soft tissue infection, or history of MRSA colonization. Aminoglycosides (gentamicin, tobramycin) may be considered as an addition to standard monotherapy in the critically ill patient with concern for resistant gram-negative bacteria. Antivirals and antifungals should be considered in patients with persistent fever or clear signs of active infection (e.g., herpetic lesions or *Candida esophagitis*) but are often outside the scope of emergency department empiric treatment.<sup>1,3</sup>

As the treatment of cancer trends more toward outpatient therapy, neutropenic fever will become a far more common occurrence in the emergency department. As the front line of evaluation and treatment for these patients, it is imperative that emergency medicine physicians readily diagnosis and manage neutropenic fever aggressively but appropriately in consultation with infectious disease and hematology/oncology providers. Early treatment with targeted antibiotic regimens, determined by individual clinical presentation along with institutional antibiograms and protocols, will help to more effectively treat neutropenic fever while minimizing the cost and risks of bacterial resistance that is often associated with broad-spectrum IV antibiotics.

## KEY POINTS

- Recognize neutropenic patients with fever and/or signs of infection, obtain pan-cultures, and treat early with antibiotics.
- Low-risk patients: Discuss with patient/oncologist the option of outpatient therapy with oral antibiotic regimen (ciprofloxacin AND amoxicillin/clavulanic acid OR clindamycin).
- High-risk patients: Start IV monotherapy with antipseudomonal  $\beta$ -lactams. For severe penicillin allergy, use aztreonam AND vancomycin or ciprofloxacin AND clindamycin.
- Vancomycin should NOT be standard empiric treatment unless there is hemodynamic instability, radiographic pneumonia, suspected vascular catheter or soft tissue infection, GPCs seen on preliminary blood culture or history of MRSA colonization.

- Viral and fungal coverage should not be routinely started in ED without evidence of active disease but should be considered in high-risk patients after discussion with consultants.

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# **MY CHEST! MY BACK! MY SICKLE CELL ATTACK!**

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**CULLEN CLARK, MD AND ANNA MCFARLIN, MD**

Pain in a patient with sickle cell disease (SCD) can be an indicator of life-threatening pathology. The provider can be lured into diagnosing and treating pain as a vaso-occlusive episode (VOE) without assessing for signs of impending clinical deterioration. A VOE is a diagnosis of exclusion; you must first rule out the emergent conditions associated with SCD by relying on a thorough history and physical exam. This chapter is not a comprehensive chapter on SCD but highlights the complications that could quickly turn a suspected VOE into a catastrophic emergency.

## **ACUTE CHEST SYNDROME**

Acute chest syndrome (ACS) is the leading cause of death in patients with sickle cell anemia.<sup>1</sup> ACS should be on the differential for any SCD patient with chest pain. Be reluctant to attribute a patient's tachypnea to pain. It is important to assess for ACS by acquiring a chest x-ray and performing a thorough physical exam; listen for rales (most common physical exam sign), decreased breath sounds, or wheezing.<sup>2</sup> Fever and cough are common symptoms of ACS.<sup>1</sup> Continuous pulse oximetry should be initiated upon arrival to the ED to assess and monitor for development of hypoxia due to ACS.<sup>3</sup>

When assessing chest pain in a patient with sickle cell anemia, keep your differential wide. These patients have a high risk for ACS, but that does not exclude them from common causes of chest pain seen in the general population such as acute myocardial infarction, pulmonary embolus, or

pneumothorax.<sup>4</sup> Rapid detection of ACS is important as early initiation of exchange transfusion and antibiotics can drastically improve outcomes.<sup>2</sup>

## **STROKE**

It is absolutely crucial to test for disability during the primary assessment of a patient in a pain episode. A VOE is not confined to the peripheral circulation; these patients are a high risk for stroke due to cell adhesion and inflammatory changes in cerebral vasculature. A thorough assessment of mental status and neurological deficit should be performed.<sup>3</sup>

Stroke is not just an adult problem. Children with SCD between 2 and 9 years of age are at significant risk for stroke.<sup>4</sup> Young children who are unable to comply with a neurologic exam, parents, or caregivers can be your best source of information regarding changes in neurologic status.

## **SEQUESTRATION**

A thorough physical exam can make a lifesaving difference. It is imperative to palpate for the patient's spleen. Splenic sequestration is equivalent to major hemorrhage; these patients lose a significant amount of blood from their intravascular volume into their spleen. The spleen is a dangerously capable reservoir for blood; it can rapidly enlarge to several times its normal size due to pooling.

Children with SCD are more likely to develop splenic sequestration as most still have adequate blood flow to the spleen. This is less common in adults with SCD because they often have splenic atrophy or fibrosis due to infarcts earlier in life.<sup>2</sup> Even with functional asplenia, adults are still at risk for hepatic sequestration.<sup>5</sup>

The initial tachycardic response to hypovolemia can be falsely attributed to pain. More specific symptoms for acute splenic sequestration include hypotension, fatigue, abdominal distention, and back pain refractory to pain medication.<sup>5</sup> A CBC will show severe anemia and thrombocytopenia with elevated reticulocyte count.<sup>4</sup> If you see these symptoms or indications of poor perfusion, palpate for splenomegaly and hepatomegaly. If no spleen is palpable but suspicion is high, consider a bedside ultrasound or abdominal x-ray to assess for splenic or hepatic enlargement.<sup>5</sup> Early detection allows for early transfusion and prevention of severe shock.



# INFECTION

Infection is a common contributory factor to the RBC sickling that triggers a VOE. As mentioned above, numerous infarcts can leave a patient functionally asplenic early in life, impairing his or her defense against encapsulated bacteria. It is crucial to determine if a VOE has been triggered by an infection. Scrutinize for signs of meningitis, osteomyelitis, and septic arthritis. In patients who present with fever or localizing signs of infection, obtain blood and urine cultures and chest x-ray, and then administer empiric antibiotics as soon as possible.<sup>3</sup>

It is important to have a low threshold for an infectious workup when treating pediatric patients under age 5 years. Younger patients may have not received all of the vaccinations that help protect from common encapsulated organisms.

There are many other serious manifestations of SCD seen in the ED. Keep aplastic crisis on the differential if the patient has a low hemoglobin. Depressed hematopoiesis due to Parvovirus B19 can be life threatening to a patient with SCD.<sup>4</sup> A patient who presents with extremity pain may be experiencing avascular necrosis, commonly mistaken for simple VOE. Lastly, young children are at risk of dactylitis caused by vaso-occlusion in the digits.

In all of these complications of SCD, early involvement of a hematologist in the treatment plan is important for patient outcome and transition of care from the emergency department.<sup>2,3</sup>

## KEY POINTS

- VOE is a diagnosis of exclusion; keep a wide differential for causes of pain in a patient with SCD.
- Initiate continuous pulse oximetry, and obtain CXR early if suspicious of ACS.
- Patients with SCD are at high risk for stroke; always assess for neurologic disability.
- Hypotension, abdominal distention, and back pain refractory to pain medication are signs of acute splenic sequestration.

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# WARFARIN REVERSAL: FACTOR IT IN

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**WILLIAM B. STUBBLEFIELD, MD AND DAREN M. BEAM,  
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Warfarin is the most widely used vitamin K antagonist (VKA) in North America for the prevention of venous thromboembolism (VTE). There are >60,000 emergency department visits for hemorrhagic complications in VKA-treated patients annually.<sup>1</sup>

Warfarin inhibits the enzyme responsible for the carboxylation of vitamin K–dependent coagulation factors II, VII, IX, X, and anticoagulant proteins C and S, making them inactive. Its dosing is guided by measurement of the international normalized ratio (INR), a standardized measurement of prothrombin time (PT), with a desired therapeutic range of 2 to 3.5. Excessive INR values occur from a host of reasons, mainly due to a large number of drugs and foods that have an effect on warfarin metabolism.

The term “reversal” for excessive INR caused by warfarin is technically incorrect. The drug is neither removed from the system nor is it bound and deterred from its mechanism of action. With treatment, we either diminish the inhibitory effect of warfarin or give more factors to replenish the functional depletion it has generated.<sup>2</sup> This is done by stopping warfarin therapy, administering vitamin K, and considering the use of fresh frozen plasma (FFP), or prothrombin complex concentrate (PCC).

In patients with modestly elevated INR without clinically evident bleeding, cessation of warfarin, careful observation, and periodic monitoring constitute the safest course.<sup>2</sup> However, the risk of clinically significant bleeding has been shown to increase when the INR is in the 3.0 to 4.5 range,

with an exponential increase in bleeding events occurring when the INR is >5.0.<sup>3</sup> Therefore, management of excessive anticoagulation from warfarin is dependent on the presence or absence of active bleeding and the degree of INR elevation ([Table 150.1](#)).

**TABLE 150.1 TREATMENT OF ELEVATED INR**

| INR     | BLEEDING              | TREATMENT   |
|---------|-----------------------|---|
| 3.5–4.9 | None                  | Hold warfarin and observe                                   |
| 5.0–9.0 | None                  | Hold warfarin and administer 1.0–2.5 mg oral vitamin K      |
| >9.0    | None                  | Hold warfarin and administer 5.0 mg oral vitamin K          |
| Any     | Uncontrolled bleeding | Hold warfarin; administer 10 mg vitamin K IV and FFP or PCC |

Vitamin K should be administered orally or intravenously with oral dosing being the preferred route of administration. In cases of excessive anticoagulation with warfarin accompanied by serious or life-threatening bleeding, vitamin K should be administered intravenously as an infusion over 20 to 30 minutes, not as a bolus.

Indications for FFP are based primarily on observational trials and expert opinion and include intracranial hemorrhage (ICH), as well as any life-threatening bleed associated with warfarin use including pericardial, retroperitoneal, or other active bleed with hypotension or a 2 g/dL drop in hemoglobin. FFP is not indicated for volume expansion, nonurgent reversal of VKA, or treatment of abnormal INR from any cause in the absence of bleeding. Administration of FFP prophylactically to nonbleeding patients is not indicated. Furthermore, limited available evidence fails to support the use of FFP in patients with an elevated INR before minor invasive procedures such as central line placement and lumbar puncture. FFP is not indicated for GI bleeds unless they meet the definition of life-threatening bleed.<sup>4</sup>

The administration of FFP can impart significant risk thus should not be given if not indicated. The associated large volume it takes to lower the INR is associated with long transfusion times, circulatory overload, risk of transfusion-related acute lung injury (TRALI), in addition to risk of pathogen transmission, allergic reactions, and hemolysis.

FFP contains all clotting factors. One unit has a volume of 200 to 250 mL. To transfuse, start with infusions of 10 to 30 mL/kg. Results will be

somewhat unpredictable; follow-up laboratory and clinical assessment will be necessary.<sup>3</sup> In general, 1 unit of FFP will increase most coagulation factors by 3% to 5% in a 70-kg adult. The common practice of administering 2 units of FFP to an adult (~7 to 8 mL/kg) will only increase coagulation factors by 10%. For a clinically relevant correction of coagulation factor deficiencies, a dose of 15 mL/kg (or 4 units in a 70-kg adult) is required. This raises most coagulation factor levels by ~20%.<sup>3</sup> FFP itself has an intrinsic INR of ~1.7, so FFP transfusion generally will not lower a patient's INR below this level. It is an error to repeatedly administer FFP to patients with a slightly elevated INR in hopes of reaching an unrealistic target INR of 1.0.

Originally developed for hemophilia B, PCC contains vitamin K–dependent coagulation factors. Octaplex contains factors II, IX, and X, and FEIBA contains II, IX, X, plus factor VII. Both contain protein C, S, and also trace amounts of heparin. The advantage is a smaller volume size and rapid delivery. This is particularly important because the majority of patients on warfarin have comorbidities where volume overload would be detrimental. A recent prospective randomized-controlled demonstrated that 4-factor PCC was superior to FFP in producing both effective hemostasis and rapid INR reduction in patients needing VKA reversal for urgent surgical or invasive procedures.<sup>5</sup>

## KEY POINTS

- Three approaches for warfarin correction depending on severity:
  - Hold warfarin.
  - Hold warfarin and give vitamin K.
  - For the severely bleeding patient, hold warfarin, give vitamin K, and replete factors.
- INR is a poor predictive metric of clinical bleeding.
- FFP is only indicated for ICH-associated and other life-threatening warfarin bleeding.
- PCC is more expensive but uses less volume and results in a faster correction
- Correction of the INR with PCC or FFP has not been shown to change morbidity or mortality.

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# EMERGENT ANTICOAGULANT REVERSAL: BE APPROPRIATELY AGGRESSIVE

**KEITH AZEVEDO, MD AND ISAAC TAWIL, MD, FCCM**

In the setting of life-threatening hemorrhage, the standard ABCs may be reconsidered as A<sup>2</sup>BC, with the additional “A” representing concomitant anticoagulant reversal. The anticoagulated patient with life-threatening hemorrhage does not allow time for nuanced discussions and thorough vetting of the evidence, as reversal is a time-sensitive action. A classic scenario of life-threatening hemorrhage requiring emergent anticoagulant reversal is that of intracranial hemorrhage (ICH). We extrapolate recommendations for other hemorrhages from this often-studied patient group. The objectives of this chapter are to: (1) explore the rationale for rapid anticoagulant reversal in life threatening hemorrhage; (2) discuss the reversal of the most common anticoagulant: warfarin; (3) identify the newer Target Specific Oral Anticoagulants (TSOACs), proposed reversal strategies, and future reversal agents in development; and (4) provide a practical reference guide that can be utilized on one’s next shift.

## ANTICOAGULANTS AND HEMORRHAGE

The most common oral anticoagulant is the vitamin K antagonist (VKA), warfarin. Increasing in popularity due to lack of required monitoring and greater anticoagulation reliability are the TSOACs, which include factor Xa inhibitors, and direct thrombin inhibitors (DTIs). Other common antithrombotic agents include antiplatelet agents. Refer to [Table 151.1](#) for mechanism of action (MOA), half-lives, and reversal. While relative safety

of all these agents has been demonstrated, a life-threatening emergency arises when these patients are bleeding, particularly in the setting of ICH. Typical ICH mortality is estimated at ~40%, increasing to ~60% when complicated by anticoagulation.<sup>1</sup> Further neurologic morbidity increases with hematoma expansion, which worsens in the setting anticoagulation.<sup>2</sup>

**TABLE 151.1 ANTITHROMBOTIC MOA/HALF-LIFE**

| ANTITHROMBOTIC AGENT                               | MOA <sup>a</sup>   | HALF-LIFE                                   |
|--|--|---|
| Warfarin   | Vitamin K antagonist/epoxide reductase inhibitor, preventing synthesis of factors II, VII, IX, X | 36 h  |
| Rivaroxaban, apixaban, and edoxaban                | Direct Xa inhibitors   | 7–21 h normal renal function                |
| Dabigatran   | Direct thrombin (IIa) inhibitor  | 14–17 h                                     |
| Aspirin  | Irreversible acetylation of COX-1 and fibrinogen   | 15–30 min, 5–7 d for full platelet recovery |
| Clopidogrel, prasugrel: thienopyridine derivatives | Irreversible inhibition of platelet ADP and platelet activity                                    | 7–8 h, 5–7 d for full platelet recovery     |

<sup>a</sup>MOA, mechanism of action.

## WARFARIN REVERSAL

Reversing anticoagulants is informed by varying levels of evidence, with warfarin reversal being the most researched. Warfarin reversal can be achieved through various agents, each differing in time of onset and duration of action: vitamin K, prothrombin complex concentrate (PCC), and fresh frozen plasma (FFP) ([Table 151.1](#)). Intravenous vitamin K has an onset of action of 2 to 6 hours and requires up to 24 hours to achieve reversal on its own. PCC is a concentrate of vitamin K–dependent factors II, VII, IX, and X, plus protein C and S as well as a small amount of heparin. Originally developed for hemophilia B treatment, PCC has been used in much of the developed world for emergent warfarin reversal due to rapid onset and reliable international normalized ratio (INR) correction. A recent randomized trial of vitamin K + PCC versus vitamin K + FFP for VKA-associated hemorrhage, compared hemostatic efficacy, and INR correction. In this industry-sponsored noninferiority study, PCC reversal was more rapid and with fewer adverse effects, which were primarily plasma-related volume overload. Thrombotic complications did not differ between groups. This



study led to FDA approval of PCC for urgent VKA reversal in the setting of major bleeding.<sup>3</sup> While there is a lack of mortality data, there are several reasons why PCC is the preferred agent over plasma. The major downside of a FFP reversal strategy is the delay to INR correction owing to the required thawing of FFP and the unpredictable INR normalization. Additional complications include transfusion-associated circulatory overload (TACO) from the colloid load and transfusion-associated lung injury (TRALI). Thus, recent AHA recommendations for VKA-associated ICH include IV vitamin K plus PCC as potentially preferable over FFP.<sup>4</sup>

## **NEWER AGENTS: TARGET-SPECIFIC ORAL ANTICOAGULANTS AND BEYOND**

These agents can be placed into two categories based on their MOA: DTI and factor Xa inhibitors. DTIs include the oral agent dabigatran and three parenteral agents bivalirudin, argatroban, and desirudin. Xa inhibitors in clinical use include rivaroxaban, apixaban, and edoxaban. Until recently, no rigorous clinical evidence existed to guide reversal of the target-specific oral anticoagulants (TSOACs), and recommendations are based on studies targeting normalization of laboratory parameters not clinically used and expert opinion. PCC may reverse these agents by activating thrombin on the platelet surface. DTIs may be cleared by hemodialysis, but this is often impractical in the emergency setting. Antidotes are currently being developed, such as the newly FDA-accepted antibody fragment idarucizumab, which showed promise in a recent observational clinical trial for reversal of dabigatran.<sup>5</sup> Concerning antiplatelet agents, consensus on reversal is less clear, but many experts assert that since these agents tend to increase bleeding, their action should similarly be reversed. While clinical trials are ongoing, recommended treatments include transfusion of functioning platelets and DDAVP (desmopressin), which induces the release of von Willebrand factor and factor VIII, augmenting hemostasis. Consider *Figure 151.1* on your next shift when a patient on anticoagulants presents with life-threatening bleeding.

## ANTITHROMBOTIC AGENTS

## REVERSAL AGENTS

### WARFARIN

- Vitamin K:  
Dose: IV 10mg  
  
PLUS EITHER
- PCC: (preferred) Less volume  
Dose: INR: 1.5-4.0: 25 u/kg  
INR: 4.0-6.0: 35 u/kg  
INR: >6.0: 50 u/kg  
Cap all doses at 100 Kg  
  
OR
- FFP: More volume  
Dose: 15-30 ml/kg

### Xa Inhibitor (Rivaroxaban)

- PCC  
Dose: 50 u/kg  
Cap dose at 100 Kg

### Direct Thrombin (IIa) Inhibitor (Dabigatran)

- Idarucizumab

### ASPIRIN

### THIENOPYRIDINE DERIVATIVES

(Clopidogrel and Prasugrel)

- Desmopressin  
Dose: 0.3 mcg/kg
- Consider Platelet  
Transfusion

**Figure 151.1**

## KEY POINTS

- Rapid anticoagulant reversal is critical in life-threatening hemorrhage.
- A warfarin reversal strategy should include 10 mg IV vitamin K plus PCC or FFP (PCC preferable).
- TSOAC reversal currently includes PCC based on limited data, and antibody fragment antidotes are currently being developed.
- While controversial, antiplatelet agent reversal includes platelet transfusion plus DDAVP.

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# **RECOGNIZE LEUKOSTASIS AND KNOW WHEN TO CONSULT FOR EMERGENT TREATMENT**

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Hyperleukocytosis is defined as a white blood cell (WBC) count exceeding 100,000/ $\mu$ L and can develop as a result of a number of hematologic malignancies and may in fact be their initial presentation. When hyperleukocytosis becomes symptomatic, it is known as leukostasis and is associated with a 1-week mortality of 20% to 40%.

Leukostasis occurs when poorly deformable blast cells become such a high proportion of the total blood volume that they increase blood viscosity and plug the microcirculation leading to reduced blood flow to the various end organs. Adding to the hypoxic damage caused by reduced blood flow, these abnormal cells have such high metabolic activity as to create areas of local hypoxemia and also may cause direct damage from cytokine release and migration into adjacent tissues.

Hyperleukocytosis and leukostasis may be seen in all types of leukemia, but there remains significant variability among those types regarding at what WBC count leukostasis is likely to develop. This is thought to be due to variability in the size and deformability of the cells. In AML, leukostasis is typically seen in patients with WBC counts of 100,000/ $\mu$ L or greater. Hyperleukocytosis is most common in CML; however, symptoms of leukostasis rarely develop during the chronic phase below WBC counts of 500,000/ $\mu$ L excepting that leukostasis can occur with the development of blast crisis. In ALL, hyperleukocytosis is common, but leukostasis is not and occurs with higher counts, typically above 400,000/ $\mu$ L. Finally, leukostasis

occurs least commonly in CLL, typically only when counts exceed 1,000,000/ $\mu$ L.

Not surprisingly, the symptoms of leukostasis correspond to those associated with local ischemia and manifest differently depending on which organ systems are most affected. Pulmonary and neurologic presentations are the most common, but any organ system can be involved with rarer presentations including myocardial ischemia, acute kidney injury, bowel infarction, acute limb ischemia, or priapism. Pulmonary symptoms include dyspnea and hypoxemia and are often associated with pulmonary infiltrates on the chest radiograph. Neurologic symptoms may span the gamut from a headache or dizziness all the way to somnolence and coma.

Patients with hyperleukocytosis are also at risk of developing other conditions which may mimic or occur concurrently with leukostasis and should be considered in the same differential. Fever commonly accompanies leukostasis, raising the specter of infection, which may be impossible to exclude in the acute setting. While a patient with hyperleukocytosis has an exceptionally high white cell count, very few of these cells are likely to be functional, creating an effective state of immunosuppression. As a result, many patients with leukostasis will be treated initially with empiric antibiotics. Similarly, while leukostasis can present with renal failure, patients with hyperleukocytosis in the absence of leukostasis are still at high risk of developing tumor lysis syndrome and subsequent renal failure. Neurologic leukostasis may be mimicked by intracranial hemorrhage, which can result from the endothelial damage and thrombocytopenia induced by hyperleukocytosis. Hyperleukocytosis in the absence of leukostasis may also have thrombotic consequences being associated with the development of disseminated intravascular coagulation (DIC). As a result, the ultimate diagnosis of leukostasis is often not a confident one, and aggressive therapy should be started at first sign of symptoms potentially attributable to leukostasis.

Hyperleukocytosis is associated with a number of misleading laboratory findings. Blood gas analysis may overestimate the severity of hypoxemia in patients with hyperleukocytosis due to hypermetabolism of WBCs in the blood sample. For this reason, pulse oximetry may provide the most useful assessment of oxygen saturation in the setting of hyperleukocytosis. The occurrence of kidney injury and tumor lysis syndrome makes the potassium level of great importance in the laboratory evaluation of the patient with hyperleukocytosis; however, the measurement of serum potassium can be complicated by the occurrence of pseudohyperkalemia in the setting of hyperleukocytosis, particularly CLL. Pseudohyperkalemia is an in vitro phenomenon in which the large number of fragile WBCs lyse in the blood

sample before plasma is removed for analysis. Using a blood gas analyzer to measure potassium in samples sent on ice can reduce the occurrence of pseudohyperkalemia.

Treatment of leukostasis primarily revolves around prompt cytoreduction while providing supportive care such as prevention of tumor lysis syndrome. Red blood cell transfusion should be avoided prior to cytoreduction when practical as this may further increase the viscosity of the circulating blood. Three basic approaches to cytoreduction can be undertaken, any of which will require oncologic consultation prior to undertaking and with the best method of initial cytoreduction being somewhat controversial. Cytoreduction has been traditionally accomplished with leukapheresis. In this method, WBCs are mechanically separated from the remainder of the blood components, which are then returned to the circulation. Alternate methods of cytoreduction include the use of oral hydroxyurea or the immediate initiation of induction chemotherapy. Hydroxyurea is given orally and reduces the WBC count by 50% to 80% within 1 to 2 days without inducing tumor lysis pneumopathy or DIC. Emergency physicians may play a role after oncologic consultation by effecting the early initiation of hydroxyurea as well as in obtaining of large bore central venous access for leukapheresis similar to that used for hemodialysis.

## KEY POINTS

- Leukostasis is associated with a high rate of mortality.
- Leukostasis is often indistinguishable from other clinical syndromes such as infection, and the diagnosis will often remain uncertain as therapy is simultaneously initiated for both conditions.
- The diagnosis of leukostasis is often uncertain, and prompt consultation for cytoreduction remains warranted.
- Hyperleukocytosis can induce spurious laboratory results including falsely decreased oxygen saturation on blood gas analysis and pseudohyperkalemia.

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# SECTION X

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## IMMUNE



## DON'T GET STUNG BY ANAPHYLAXIS

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Anaphylaxis is a serious, life-threatening, often misdiagnosed problem that requires immediate attention in the emergency department (ED) setting. The key for better outcomes is early recognition and intervention. If anaphylaxis is not recognized due to nonspecific symptoms, consequences can be fatal. Reviewing a detailed description of the events leading up to the episode is essential to diagnosis and recognition.

Anaphylaxis triggers are important to identify in the ED. In the pediatric population, the most likely triggers are food such as peanuts/tree nuts, shellfish/seafood, eggs, cow's milk, and wheat. In adults, the most likely causation is an insect sting, belonging to the hymenoptera order. Other causes include antibiotics, latex, or idiopathic.<sup>1</sup>

Experts have created three diagnostic criteria that allow clinicians to recognize the subtleties of presentation.<sup>2</sup> The diagnosis of anaphylaxis is HIGHLY likely when ONE of the following three criteria is demonstrated:

**Criterion 1:** Acute onset of symptoms involving the skin and/or mucosa PLUS either respiratory compromise or reduced blood pressure (or associated signs and symptoms of end-organ damage). This is considered the classic presentation of anaphylaxis with acute pruritus, flushing, lip/tongue swelling, hives, dyspnea, bronchospasm, stridor, hypoxemia, syncope, and mental status changes.

**Criterion 2:** The second criterion is more subtle. The patient will have two or more of the following symptoms after exposure to a

**suspected allergen:** skin/mucosal tissue involvement, respiratory compromise, reduced blood pressure (or associated symptoms), and/or persistent GI symptoms. The patient may not have skin involvement. It is important to note that there will be no skin symptoms in 20% of cases of anaphylaxis.

**Criterion 3:** Requires exposure of a **known allergen** to the patient. The only symptom in this category is reduced BP, in adults less SBP <90 mm Hg (or 30% decrease from baseline if known). For children under 10 years of age, hypotension is defined as  $70 \text{ mm Hg} + 2 \times \text{age}$ . Clinically, the patient may present with syncope as a sign of hypotension. It is important to remember this category, as it can often be missed.<sup>3</sup>

Administration of epinephrine should be limited to patients who meet the aforementioned criteria. In rare instances, epinephrine should be given when none of these are fulfilled. An example would be if someone with a history of a near-fatal reaction to an allergen presented to your ED with pruritus, urticaria, and flushing.

Death from anaphylaxis will usually occur quickly secondary to airway obstruction and cardiovascular collapse. A patient's sense of impending doom (*angor animi*) is worrisome and should never be ignored. Asthmatics are at increased risk of fatal anaphylaxis due to delay in diagnosis if symptoms are mistaken for asthma.<sup>4</sup>

Review your patients' medications. Antihypertensive medications including  $\beta$ -blockers, ACE inhibitors, and  $\alpha$ -blockers may decreased the response to epinephrine and, in the case of  $\beta$ -Blockers, may require glucagon administration with treatment of the anaphylaxis.<sup>1</sup>

History and symptoms always trump the need for laboratory testing; rapid treatment should always be the priority. After onset of symptoms, plasma tryptase will remain elevated for 3 hours and plasma histamine less than 60 minutes. These tests may have utility for the allergist on follow-up.<sup>5</sup>

Always remember ABCs. Immediate management should include removal of the inciting agent, IM epinephrine, placement of the patient in the supine position to prevent empty ventricle syndrome, supplemental O<sub>2</sub> (8 to 10 L by facemask), and volume resuscitation. Hypotensive patients should receive 1 to 2 L of normal saline at 5 to 10 mL/kg for adults and 20 mL/kg for children over 5 to 10 minutes for cardiovascular support. The airway should be secured without delay for signs of stridor or significant respiratory distress.

Intramuscular injection is the preferred modality for delivery of

epinephrine; it will allow for a more rapid increase in the tissue concentrations of the drug. An initial dose of 0.3 to 0.5 mg/dose of the 1:1,000 concentration should be injected into the midouter thigh. This can be repeated in 5 to 15 minutes based upon the clinical picture. For children and infants, the dose is 0.01 mg/kg with a maximum dose of 0.5 mg. If a patient has persistent symptoms despite multiple doses of IM epinephrine, a continuous intravenous infusion should be started (2 to 10 mcg/min for adults, 0.1 to 1 mcg/kg/min for children). Epinephrine should always be instituted immediately based upon clinical symptoms.<sup>5</sup>

Adjunctive medications such as H1/H2 antihistamines, bronchodilators, and glucocorticoids have not been found useful for the immediate systemic treatment of anaphylaxis. These adjunctive therapies may provide symptomatic relief for itching or wheezing. Glucocorticoids may be given at discharge for 3 days, as most cases of biphasic reactions will occur within 72 hours. These should never be used as first-line therapy.<sup>2</sup>

Patients who require multiple doses of epinephrine or those with severe symptoms including hemodynamic instability should be admitted for observation. All others can likely be safely discharged after an observation period in the ED of 4 to 6 hours. Patient education prior to leaving your ED is of the utmost importance due to the possibility of biphasic anaphylaxis or re-exposure to the allergen. The patient should be instructed to IMMEDIATELY return to the ED if any of the diagnostic symptoms recur within this time period. The patient should be discharged only after EpiPen training, receiving an EpiPen, and referral to an allergist.<sup>5</sup>

## KEY POINTS

- Immediate management of anaphylaxis should include ABCs, removal of the inciting agent, IM epinephrine, supplemental O<sub>2</sub>, and volume resuscitation.
- There is no absolute contraindication to the administration of epinephrine in suspected anaphylaxis.
- Adjunctive medications have not been found useful for the treatment of immediate anaphylaxis.
- Asthmatics are at increased risk of fatal anaphylaxis due to delay in diagnosis.

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## ANGIOEDEMA AND ANAPHYLAXIS ARE NOT THE SAME, THEY JUST HAPPEN TO PRESENT SIMILARLY

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LUI CALEON, MD, MPH AND PIERRE DETIEGE, MD

The differential for a patient presenting to you in the emergency department with swollen lips may include overzealous plastic surgery but is more likely to be due to either an allergic reaction or medication side effect. Acute tissue swelling is caused by multiple pathways. Ruling out a potential life-threatening process is crucial. Angioedema and anaphylaxis present similarly, are commonly confused for each other, but have differing pathophysiology and significantly different treatments.

Simply, anaphylaxis is an acute allergen-mediated reaction, while angioedema is a vascular reaction. More specifically, anaphylaxis is a true systemic hypersensitivity IgE-mediated allergic inflammatory reaction. Angioedema, however, is considered a noninflammatory disease state during which intravascular fluid extravasates secondary to increased capillary permeability into the dermis or submucosa, most commonly in the face, upper airway, and gastrointestinal tract. This vascular reaction results in a deep well-demarcated and asymmetrical nonpitting edema in the subcutaneous dermis thought to be similar to the more superficial wheal-and-flare-type reaction seen in allergic urticaria. Angioedema is most commonly idiopathic but can be ACE inhibitor induced, hereditary or acquired with C1-esterase deficiency. Anaphylaxis on the other hand is most commonly associated with adverse drug reactions and insect stings in adults and food hypersensitivities in children. Both angioedema and anaphylaxis can be life threatening ([Table 154.1](#)).

**TABLE 154.1 RISK FACTORS TO DEVELOPING CLINICALLY SIGNIFICANT ANGIOEDEMA**

|   |
|---|
| ACE inhibitor–induced cough   |
| African American race   |
| Age >65   |
| Female sex  |
| Previous hx of angioedema   |
| Previous hx drug-related rash or seasonal allergies                           |
| Preexisting narrowed esophageal space (obesity, previous surgery trauma, OSA) |

The mechanism for idiopathic (spontaneous) angioedema is not well understood. ACE inhibitor angioedema however, while rarer with an incidence of 0.1% to 0.7% in patients on pharmacotherapy, accounts for upward of 30% of cases seen in the emergency department. Angiotensin is a potent hormone that results in vasoconstriction and the stimulation of aldosterone causing blood pressure elevation. Bradykinin increases capillary permeability and is a potent vasodilator considered to be 10 times more effective than histamine. Angiotensin converting enzyme inhibitors, or ACE inhibitors for short, are used widely in the treatment of hypertension by blocking the conversion of angiotensin I to angiotensin II consequently blocking bradykinin degradation. The subsequent accumulation of bradykinin most commonly results in bronchospasm, which results in a dry irritating “hacking” cough. The deposition of surplus bradykinin into airway and GI tissue is thought to precipitate clinically significant angioedema. The onset of ACE inhibitor–induced drug side effects typically occur within the first week of drug initiation, but complications have been documented upward of 7 years after being prescribed pharmacotherapy.

Diagnosis and differentiation of both angioedema and anaphylaxis are made clinically based upon the patient’s presenting symptoms, situational context, and prior medical history. Imaging and bloodwork are largely unnecessary and delay proper treatment. Histamine and tryptase levels may be elevated in the setting of anaphylaxis only (not angioedema) but are extremely time sensitive and are “send out labs” that won’t be available to assist in emergency clinical decision making. Patients with multiple chronic presentations may be candidates for bloodwork to assist outpatient diagnosis if their symptoms began 10 to 90 minutes prior to blood draw.

Angioedema is often asymmetrically distributed in areas that are not gravity dependent. Patients may present with hallmark lip and lingular

swelling or with more vague complaints such as abdominal pain from bowel wall edema. In contrast, anaphylaxis presents more broadly. Worrisome signs that may indicate possible need for airway intervention are voice change, hoarseness, dyspnea, or stridor.

Angioedema is self-limited. In the absence of airway compromise, localized swelling will resolve in 24 to 72 hours after the immediate cessation of the offending agent (in most cases ACE inhibitor discontinuation). Although not specifically studied or proven, adjunct pharmacotherapy with H1/H2 antihistamines and glucocorticoids is generally recommended, especially in cases of diagnostic uncertainty. In the event of airway compromise or systemic hypotension, IM epinephrine is associated with a marked decrease in mortality. Adjunct pharmacotherapy may decrease length of illness but has not been shown to affect mortality. Classically, ACE inhibitor-related angioedema is refractory to pharmacotherapy. Current FDA-approved treatment of angioedema includes the use of fresh frozen plasma, C1 esterase concentrate, or newer agents such as recombinant C1 inhibitors, kallikrein inhibitors, or bradykinin receptor antagonists. Securing the patient's airway "sooner rather than later" is recommended, and having a lower threshold to intubate is advised ([Table 154.2](#)).

**TABLE 154.2 RESCUE TREATMENT**

|   | <b>ADULT DOSING</b> | <b>PEDIATRIC DOSING</b> |
|---|---------------------|-------------------------|
| IM epinephrine (1:1,000) to thigh every 15–20 min | 0.3–0.5 mg          | 0.03 mg/kg (max 0.5 mg) |
| Oxygen  |                     |                         |
| Initial normal saline bolus                       | 1–2 L               | 20 mL/kg                |
| Diphenhydramine IV if skin involvement            | 25–50 mg            | 1–2 mg/kg (max 50 mg)   |
| Ranitidine IV                                     | 50 mg               | 1 mg/kg (max 50 mg)     |
| Methylprednisolone IV                             | 125 mg              | 1–2 mg/kg (max 125 mg)  |

Prior to discharge, patients should be advised to avoid any suspected offending agents that may have precipitated their illness. If used for stabilization, patients should also be prescribed a short course of antihistamines and steroids to prevent decompensation from any prolonged or rebound episodes. Provide return instructions regarding symptoms that may be associated with recurrence. If you have any concerns about airway involvement, admit the patient to a monitored unit for observation ([Table 154.3](#)).



### TABLE 154.3 PROPOSED ANGIOEDEMA HOSPITAL DISPOSITION BY STAGING

**STAGE I:** *Facial rash/edema, lip edema:* outpatient management

**STAGE II:** *Soft palate edema:* outpatient vs. floor admission

**STAGE III:** *Lingular edema:* ICU monitoring

**STAGE IV:** *Laryngeal edema:* ICU monitoring

## KEY POINTS

- While most adverse drug reactions to ACE inhibitors occur in the first week of treatment, angioedema can present many years later. Immediately discontinue ACE inhibitors in all patients with signs of angioedema.
- Most major adverse events related to airway interruption secondary to angioedema and anaphylaxis occur in the first 2 hours from symptom onset. Observe patients for 4 to 6 hours after initial symptom onset (not necessarily from ED presentation).
- Both angioedema and anaphylaxis are diagnosed clinically. Labs are generally considered unnecessary.
- Secure the patient's airway for any worrisome signs including voice change, hoarseness, dyspnea, and stridor.

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# TREACHEROUS TRANSPLANT TOXICITIES

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**CHRISTOPHER J. EDWARDS, PHARM D, BCPS AND TSZ  
YEE  
(JANICE) TSUI, PHARM D, BCPS**

The CDC reports that there are 700,000 ED visits for adverse drug events each year in the United States. Chronic immunosuppressive regimens following solid organ transplant present a unique challenge given their diverse array of adverse effects, medical necessity, and narrow therapeutic index. This chapter will attempt to familiarize the reader with toxicities of common immunosuppressants by presenting a chief complaint that could be explained by the medication discussed in the following section.

## CALCINEURIN INHIBITORS

*“I’ve had this weird tremor for a while, plus this terrible  
headache for 2 weeks”*

The calcineurin inhibitors (CNIs) cyclosporine and tacrolimus (aka Prograf) revolutionized solid organ transplant in the early 1980s and remain widely used today. Aptly named, these agents disrupt the intracellular process responsible for T-cell activation by inhibiting calcineurin.<sup>1</sup> CNIs have also been used in the management of various autoimmune diseases, although their utility for nontransplant indications is limited by their significant toxicities.

### ***Neurotoxicity—Shakes and Headaches***

Most patients will eventually develop tremor while on a CNI.<sup>1</sup> Headaches are also fairly common. Psychiatric effects including restlessness, disorientation, and visual hallucinations can occur, as can changes in speech articulation. More severe conditions such as extrapyramidal syndrome, seizures, status epilepticus, encephalopathy, and posterior leukoencephalopathy have been associated with both CNIs. CNS effects are more common shortly after initiating CNIs, but can occur months or even years into therapy. While serum drug concentrations should be measured in patients experiencing CNS effects, there is not a strong correlation between drug levels and neurotoxicity.

### ***Nephrotoxicity—Job Security for Transplant Surgeons***

Despite their frequent use in renal transplant, both cyclosporine and tacrolimus are inconveniently nephrotoxic.<sup>1</sup> Functional abnormalities such as vasoconstriction of the afferent arterioles, activation of the renin-angiotensin-aldosterone system, mitochondrial pore blockage, and endothelial dysfunction are thought to contribute to structural abnormalities such as arterial hyalinosis, tubular atrophy, glomerulosclerosis, and isometric vacuolization of tubular cells that are commonly seen with long-term exposure to CNIs. Therapeutic drug monitoring is essential given that both high and low serum drug concentrations have been associated with CNI-induced nephrotoxicity.

### ***Other Toxicities***

Cardiovascular, metabolic, and electrolyte derangements can also occur. Both tacrolimus and cyclosporine can cause these effects; however, dyslipidemias, hypertension, and hyperuricemia are more common with cyclosporine while diabetes is more frequently associated with tacrolimus.

### ***Pharmacokinetics and Levels***

Both tacrolimus and cyclosporine undergo extensive hepatic metabolism via CYP3A4 and are primarily eliminated in the feces. Target serum levels depend on many factors including concomitant immunosuppressant use, induction agents used, and time from transplantation.

## **MYCOPHENOLATE**

*“I’ve been pooping like crazy, and came in when I saw*

*blood”*

Mycophenolate, aka CellCept or MMF, is commonly used for the prevention and treatment of acute rejection for a variety of solid organ allografts.<sup>2</sup> Mycophenolate is a noncompetitive reversible inhibitor of inosine monophosphate dehydrogenase. By interfering with this pathway, DNA synthesis of lymphocytes is inhibited.

### ***Gastrointestinal—A Pain in the Gut***

The most common side effects from mycophenolate are gastrointestinal, including diarrhea, abdominal pain, and dyspepsia.<sup>2,3</sup> While less common, patients can develop serious effects including rectal hemorrhage, duodenal ulcers, erosive enterocolitis, and colon perforation.

### ***Hematologic—That CBC is MMF’d!***

Mycophenolate inhibits DNA synthesis of lymphocytes and other blood cells. More than 10% of patients taking mycophenolate experience leukopenia.<sup>3</sup> Other hematologic side effects include anemia, thrombocytopenia, pancytopenia, and agranulocytosis.

### ***Infection—An Unsurprising Consequence***

Prolonged immunosuppression with mycophenolate increases the risk of opportunistic infections. More than 15% of patients may experience cytomegalovirus (CMV) viremia or tissue-invasive disease.<sup>2</sup> Other common infections include herpes simplex virus and *Candida*.

### ***Pharmacokinetics and Levels***

Mycophenolic acid (MPA) is metabolized to MPA glucuronide in the liver and is ultimately excreted via the kidneys.<sup>2</sup> Routine monitoring of mycophenolate levels is uncommon, as levels are not correlated with an increase in adverse effects.

## **SIROLIMUS**

*“I’ve had this cough for a while and now my legs are all swollen”*

Sirolimus (aka rapamycin) is a macrolide antibiotic that was originally investigated for its antifungal activity.<sup>4</sup> During preclinical investigation, sirolimus was shown to possess immunosuppressive activity by blocking the mammalian target of rapamycin (mTOR) receptor, which subsequently inhibits T-lymphocyte activation and antibody production. It is used mainly following renal transplantation.

### ***Hematologic—Platelets, RBCs, WBCs...None are safe***

The most common side effects of sirolimus are a dose-dependent decrease in platelets and a non-dose-dependent reduction in white blood cells.<sup>4</sup> As a result, sirolimus carries a black box warning regarding increased risk of infection. Sirolimus has also been associated with anemia, thrombocytopenia, and epistaxis.

### ***Pneumonitis—One Tough SOB***

In a retrospective review of 186 liver transplant patients, 2.2% of patients developed pneumonitis attributed to sirolimus.<sup>4</sup> The most common presenting symptoms were dyspnea, cough, and fatigue. Resolution of sirolimus-induced pneumonitis occurred upon cessation of therapy; however, recovery can take several months.

### ***Other Toxicities***

Patients taking sirolimus can have significantly increased cholesterol and triglyceride levels. Other common side effects (>30%) include peripheral edema, abdominal pain, headache, fever, urinary tract infection, nausea, and arthralgia.<sup>4</sup>

### ***Pharmacokinetics and Levels***

Sirolimus is mainly metabolized by CYP3A4 in the liver and eliminated via fecal route.<sup>4</sup> Medications that inhibit CYP3A4 enzymes such as diltiazem and ketoconazole may increase sirolimus levels. Trough levels above 15 ng/mL are correlated with an increased risk of hypertriglyceridemia, thrombocytopenia, and leukopenia.

## **KEY POINTS**

- Levels *might* be helpful for cyclosporine, tacrolimus, and sirolimus, but not mycophenolate.
- Patients are often on multiple immunosuppressant medications, increasing the risk for additive toxicities.
- A good medication history on transplant patients can go a long way.

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# THINK OUTSIDE THE “GRAFT BOX” WHEN EVALUATING THE TRANSPLANT PATIENT

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**TIMOTHY S. DAVIE, MD**

After finishing your training, you decided you'd take a break from all the complicated tertiary-care patients and head out to a small rural area to practice bread-and-butter emergency medicine. One quiet evening you enter the room of your next patient, a 44-year-old businessman traveling cross country for a convention. On day 2 of his road trip, he started having some deep, achy right lower abdominal pain, low-grade fever, and anorexia. You're ready to click “sign” on your order for contrasted CT to confirm suspected appendicitis when he tells you that 8 months prior, he received a kidney transplant from his sister because of his worsening hypertensive nephropathy. His graft is in the right iliac fossa, and the surgeon left his appendix in place. You quickly realize that this isn't going to be as quick a case as you thought.

The most important first step in the approach to the transplant patient is to take a step back to avoid premature closure. Don't forget to consider emergent possibilities explained by things other than the graft. In the patient above, appendicitis is as much a “can't miss diagnosis” as it would be in the nontransplant patient. Ensure that your history and exam are sufficiently thorough to cover all your bases. Next, develop a plan to obtain preliminary diagnostic information, as well as a plan to discuss your findings and proposed treatment plan with the patient's transplant surgery team. Remember that many of your diagnostic and therapeutic maneuvers can be harmful to the precious graft.

Once you've considered non-transplant-related problems, consider graft infection. Rejection accounts for only 6% of transplant-related diagnoses in the ED, while infection accounts for 36%. These patients are on lifelong medicines to suppress the immune system. In addition, nonnative organs are inherently more susceptible to infection even in the absence of immune-suppressing drugs. If you see leukocytes in the urine of a renal transplant patient, don't automatically assume that this confirms infection, as rejection alone can also give rise to this finding.<sup>3</sup>

Graft rejection is the leading cause of death in the first year after transplant. Assume rejection only after you've considered graft infection, as well as non-transplant-related emergencies. In essence, rejection is a diagnosis of exclusion. Allow your mind to settle on it only after you've thought about perigraft abscess, obstruction, vascular stenosis, and thrombosis. Remember that many of the antirejection medications themselves are nephrotoxic, so you also need to differentiate between primary acute rejection and drug-induced nephrotoxicity. Luckily, with the advent of newer immunosuppressants, the incidence of rejection during the first posttransplant year has dropped from 40% to 50% in the past to 15% to 25%.<sup>1</sup> When ordering labs, don't forget to check drug levels of those immune suppressives. Before pulling the trigger on the CT, carefully consider the risk and benefit of nephrotoxic intravenous contrast dye. Involve the transplant team early.

Once you've developed a preliminary diagnosis, do everything possible to contact the transplant surgery team responsible for the patient. If your patient is sick and you can't contact a transplant team, seriously consider transfer to a referral center who will be able to do so. If steroids are recommended as part of the treatment course, give them slowly (over 60 to 120 minutes), as sudden death by cardiac arrhythmias have been attributed to rapid administration of steroids in transplant patients.<sup>1</sup> In most infectious problems, two or more antibiotics are indicated. Ensure that you discuss this carefully with the transplant team, as various antibiotics, including aminoglycosides, are inherently nephrotoxic as well.<sup>2</sup>

After a more thorough history and exam of your patient, you learn that his transplant surgeon is someone you went to medical school with. You obtain labs, which show a creatinine of 2.3, up from the patient's baseline of 1.2. His tacrolimus level is within normal limits. You contact your old classmate, and after briefly reminiscing over the good old days, he recommends the patient get a noncontrast CT and be started on high-dose steroids, methylprednisolone, 1,000 mg daily for 3 days. The radiologist is able to adequately rule out appendicitis despite absence of contrast



enhancement, and the patient is transferred to the nearby university medical center to continue his treatment. He is discharged 3 days later, in time to catch a flight and make the last 2 days of his conference. You receive a thank-you card in the mail a week later.

## KEY POINTS

- Do not anchor on graft-related problems without considering other possibilities.
- Graft infection is much more common than is graft rejection; consider it first.
- Always contact the transplant surgeon or coordinator before deciding disposition.
- Always give steroids as slow infusion rather than rapid bolus.
- Do not use IV contrast, NSAIDs, nephrotoxic drugs (e.g., aminoglycosides, amphotericin-B), or drugs metabolized by the P-450 system (e.g., diltiazem, conazoles, azithromycin, phenytoin) without consulting a transplant physician.
- Do not forget to obtain immunosuppressive drug levels.
- Do not forget to order fungal cultures in addition to regular blood cultures in the infected transplant patient.

## ACKNOWLEDGMENT

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# DO'S AND DON'TS FOR MANAGING HEART TRANSPLANT PATIENTS IN THE ED

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LAWRENCE DELUCA JR., EdD, MD

**1) Do remember three principal diagnoses: Rejection, Infection, Everything Else!**

Transplant patients can present with a variety of illnesses beyond what their age group and comorbidities suggest. Due to immunosuppression, heart transplant patients can present with vague, atypical signs and symptoms. Patients in rejection may present with congestive heart failure (CHF).

**2) Do a complete H&P, including transplant-related issues.**

Focus not only on the chief complaint but also on current transplant regimen and any medication/compliance issues. Examine the patient for signs of CHF or volume overload. Screening ECG and chest x-ray are almost always warranted. Consider bedside ECHO to help assess volume status, cardiac function, and response to treatment.

**3) Don't call the general surgeon before the cardiac surgeon!**

Beware the heart transplant patient with vague abdominal or right upper quadrant pain. Consider CHF and hepatic congestion as a cause of symptoms before you rush to have their gallbladder plucked out.

**4) Do call the transplant coordinator (TC)!**

If the patient cannot provide a history, the TC is your lifeline. TCs can help shed light on prior transplant-related complications or compliance issues. TCs can help arrange transfer to their home center for further management or routine follow-up after ED discharge. TCs can tell you which

drug levels to assess and when to assess them.

**5) Don't do chest compressions on a patient who has a ventricular assist device (VAD)!**

When an end-stage heart failure patient has a VAD, malfunction is rarely the cause of the arrest, and chest compressions risk fatally dislodging the device. Provide standard ACLS measures (antiarrhythmics, pressors, etc.) and supportive care.

**6) Do assess volume status during resuscitation!**

The transplant patient in shock can be challenging and confusing. Oversuppressed patients are prone to septic shock. Chronic diuretic therapy and acute nausea and vomiting make patients prone to hypovolemia, and carefully titrated resuscitation can optimize intravascular volume. However, if the patient is fluid overloaded, he or she may be in cardiogenic shock, and additional fluid may worsen vascular congestion.

Common ED methods of assessing volume status and fluid responsiveness include measurement of CVP, measuring IVC diameter with ultrasound, and passive leg raise (PLR). While the CVP/volume status relationship has been questioned in recent literature, a severely elevated CVP is strongly suggestive of right heart failure, suggesting additional fluid would be deleterious.

A fluid challenge (250 to 500 mL) is the most direct means of assessing volume responsiveness and treating volume depletion. For patients who may not tolerate much volume, try a PLR. Starting with the patient's legs down and head of bed at 45 degrees, place the patient with the head flat and raise the legs 45 degrees. If HR decreases 10 to 20 bpm and/or SBP increases by 10 to 20 mm Hg, the patient will likely be fluid responsive. PLR represents a transient autoinfusion of ~250 mL that it is completely reversible—if the patient doesn't tolerate the maneuver, put him or her upright again.

**7) Do support blood pressure, but Don't worsen cardiogenic shock!**

In HYPERTensive patients with signs of CHF, consider preload and afterload reduction. Nitroglycerin can be started at 10 to 20 mcg/min and titrated to effect. Nitroprusside can also be used. Avoid beta-blockers in acute cardiogenic shock.

HYPOTensive patients are a bit more dicey. In the hypotensive patient, there is generally an abnormality of vascular tone, whether compensatory (as in vasoconstriction in hypovolemia) or part of the problem (vasoplegia in septic shock). Crude assessment is via extremity temperature—"cold shock" (vasoconstriction) versus "warm shock" (vasodilatation). Hypotensive patients who are not fluid responsive will require vasopressor or inotropic

support.

Pressors can increase vascular tone, making cardiogenic shock worse. Change your management if your patient fails to improve with (or gets worse with) pressors! The American Heart Association recommendations for management of cardiogenic pulmonary edema follow:

SBP < 70 + signs of shock—Start NOREPinephrine.

SBP 70 to 100 + signs of shock—Start DOPamine.

SBP 70 to 100, no signs of shock—Start DOBUTamine.

SBP > 100—Start NITROglycerin or nitroprusside for afterload reduction.

### **8) Don't be afraid to start inotropes!**

Assess the three main determinants of oxygen delivery: cardiac output (HR × stroke volume), hemoglobin concentration, and oxygen saturation. Correct hypoxemia and anemia to keep Hb at least 8 g/dL. Get a VBG and evaluate ScvO<sub>2</sub>% (normal is around 70% to 75%). Lower ScvO<sub>2</sub>% values could be due to hypoxia, anemia, or low cardiac output. If you've corrected hypoxia and anemia and your patient still looks shocky, initiate inotropic support. Start dobutamine at 5 mcg/kg/min (or milrinone at 0.25 mcg/kg/min). Signs of adequate therapy are improvement in shock, increase in ScvO<sub>2</sub>%, and normalization of lactate.

### **9) Do use positive pressure ventilation (PPV) for acute pulmonary edema and refractory shock!**

PPV benefits patients with pulmonary edema or cardiogenic shock by reducing work of breathing, decreasing preload (by increasing intrathoracic pressure), increasing myocardial transmural pressure (facilitating ventricular emptying during systole), and, as a “thoracic pump,” augmenting cardiac flow. Positive end-expiratory pressure (PEEP) can help alveolar recruitment.

For patients with normal mental status and airway reflexes, BiPAP is useful in exacerbations of CHF or COPD. Respiratory failure due to other causes may require endotracheal intubation.

If trialing BiPAP, be sure to assess the patient frequently. If the patient is not substantially improved within the first hour, or gets worse, intubate without delay.

### **10) Don't hesitate to transfer critically ill patients to their transplant center.**

Working with the TC, early stabilization, and transfer may be the safest and most appropriate option for a heart transplant patient.

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# ANAPHYLAXIS AND EPINEPHRINE— CAN YOU HAVE ONE WITHOUT THE OTHER?

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**RONNA L. CAMPBELL, MD, PhD**

Anaphylaxis is a potentially fatal allergic reaction that is most commonly treated in the emergency department (ED). Epinephrine is the drug of choice for the treatment of anaphylaxis, and studies have shown that delayed epinephrine administration is associated with increased mortality. In addition, ED studies of anaphylaxis have demonstrated that anaphylaxis is often undiagnosed and undertreated. Furthermore, it has been shown that many ED anaphylaxis patients were not prescribed self-injectable epinephrine.

Do all patients with anaphylaxis need to be treated with epinephrine and subsequently prescribed self-injectable epinephrine? Are there ever times when a patient experiencing an allergic reaction that does not meet anaphylaxis diagnostic criteria should be treated with epinephrine? Before answering these questions, we first need to understand how to diagnose anaphylaxis.

Anaphylaxis clinical presentations can be widely varied, and not all cases include cutaneous manifestations or multiple organ systems. The National Institutes of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network (NIAID/FAAN) criteria may be helpful in the diagnosis of anaphylaxis ([Figure 158.1](#)). These criteria are 97% sensitive and 82% specific. Thus, they are helpful but cannot replace clinical judgment.

Anaphylaxis is highly likely when any one of the following 3 criteria are fulfilled:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g. generalized hives, pruritus or flushing, swollen lips-tongue-uvula)

AND AT LEAST ONE OF THE FOLLOWING

- a. Respiratory compromise (e.g. dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
- b. Reduced BP or associated symptoms of end-organ dysfunction (e.g. hypotonia [collapse], syncope, incontinence)

2. Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours):

- a. Involvement of the skin-mucosal tissue (e.g. generalized hives, itch-flush, swollen lips-tongue-uvula)
- b. Respiratory compromise (e.g. dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
- c. Reduced BP or associated symptoms (e.g. hypotonia [collapse], syncope, incontinence)
- d. Persistent gastrointestinal symptoms (e.g. crampy abdominal pain, vomiting)

3. Reduced BP after exposure to known allergen for that patient (minutes to several hours):

- a. Infants and children: low systolic BP (age specific) or >30% decrease in systolic BP\*
- b. Adults: systolic BP of <90 mm Hg or >30% decrease from that person's baseline PEF, Peak expiratory flow; BP, blood pressure.

\*Low systolic blood pressure for children is defined as <70 mm Hg from 1 month to 1 year, <(70 mm Hg + [2 × age]) from 1 to 10 years, and <90 mm Hg from 11 to 17 years.

Modified from Sampson et al, (2006). Used with permission.

**Figure 158.1** NIAID/FAAN clinical criteria for diagnosing anaphylaxis.

Nevertheless, understanding the NIAID/FAAN criteria will help you recognize atypical anaphylaxis. Anaphylaxis should be suspected in any patient who meets one of the three criteria. All patients must have a rapid onset of symptoms. To meet criterion #1, the patient must have a cutaneous manifestation along with respiratory OR cardiovascular organ system involvement. To meet criterion #2, the patient would need to be exposed to a likely trigger and have involvement of two or more organ systems (cutaneous, respiratory, cardiovascular, or gastrointestinal). To meet criterion #3, the patient needs to have an exposure to a known trigger along with hypotension.

A few things are important to note with regard to the diagnosis of anaphylaxis. First, although anaphylaxis can cause shock, a patient does not need to have shock to have anaphylaxis. Second, although an anaphylactic reaction will typically involve more than one organ system, it is possible that a patient could have hypotension as the only presenting symptom (such as a

patient that experiences syncope after a bee sting and presents with hypotension). Third, it is important to recognize mild anaphylaxis presentations (such as a patient with a rash and vomiting after a peanut exposure). Recognition of mild presentations provides an opportunity to prevent potential progression to more serious symptoms and also to prevent future anaphylactic reactions and possible fatality by educating the patient and prescribing self-injectable epinephrine. Studies of fatal and near-fatal anaphylaxis have shown that most of these patients did not have a history of severe reactions.

Now, we can talk about epinephrine. As with any life-threatening medical condition, anaphylaxis management starts with A, B, C's, IV, O<sub>2</sub>, and monitoring. The decision to administer epinephrine is based on clinical judgment. There are no absolute contraindications for epinephrine administration in the setting of anaphylaxis, and epinephrine is very safe when administered appropriately. Some patients may need epinephrine even if they don't meet the NIAID/FAAN criteria for anaphylaxis. For example, if a patient with a history of severe anaphylaxis due to a peanut allergy presented with diffuse urticaria following an exposure to a peanut, epinephrine should be promptly administered to prevent symptom progression. In contrast, due to the frequently self-limited nature of anaphylaxis, a patient presenting several hours after an anaphylactic reaction with symptoms that initially met NIAID/FAAN criteria may no longer require administration of epinephrine if the symptoms have resolved. Thus, when it comes to acute anaphylaxis and epinephrine ADMINISTRATION, you CAN have one without the other.

In contrast, after the acute management of anaphylaxis, ALL patients should be PRESCRIBED self-injectable epinephrine, even if they did not require administration of epinephrine. Furthermore, patients who may be at risk of future anaphylaxis should also be prescribed self-injectable epinephrine even if the current reaction did not meet criteria for anaphylaxis. Ideally, patients should be provided with a prescription for two autoinjectors because ~5% to 15% of patients will require more than a single dose of epinephrine during anaphylaxis management. Therefore, when it comes to anaphylaxis and prescribing self-injectable epinephrine, you CAN'T have one without the other.

## KEY POINTS

- The diagnosis of anaphylaxis is based on clinical judgment, but



understanding the NIAID/FAAN criteria will help identify atypical anaphylaxis presentations.

- Patients don't need to have anaphylaxis to need epinephrine.
- Not all patients with anaphylaxis will need epinephrine (if symptoms have resolved).
- Prescribe self-injectable epinephrine for all patients with anaphylaxis or who may be at risk of future anaphylaxis.

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# **SECTION XI**

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# **INFECTIOUS DISEASE**

## AVOID RELYING ON THE PRESENCE OF SIRS TO DIAGNOSE SEPSIS

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KAMI M. HU, MD AND JOSEPH P. MARTINEZ, MD

Sepsis is defined as life-threatening organ dysfunction due to a dysregulated host response to infection. In the United States, sepsis is the tenth overall cause of death and the number one cause of death in noncoronary intensive care units. Current mortality rates from sepsis range from 20% to 60%. Prompt recognition and treatment of sepsis is imperative to prevent shock and limit the high morbidity and mortality associated with the diagnosis.

Since the definitions of sepsis were first proposed by the Consensus Conference of American College of Chest Physicians (ACCP) and the Society of Critical Care Medicine (SCCM) in 1991 ([Table 159.1](#)), the human body's systemic response to infection has been considered a continuum of worsening severity beginning with the systemic inflammatory response syndrome (SIRS) and progressing to organ dysfunction and refractory hypotension. Importantly, SIRS is not specific to infectious disease; patients with burns, severe pancreatitis, trauma, and other inflammatory illnesses often meet the definition of SIRS. Similarly, while some of the SIRS criteria are clear indicators of possible infection (i.e., fever, leukocytosis), less obvious criteria such as tachypnea or leukopenia do not always make an infection readily apparent. SIRS criteria alone perform poorly with regard to diagnosis of infection, with a sensitivity of 70% and specificity of 35% in emergency department patients with at least two of the four criteria. Also, limiting the diagnosis of SIRS to two or more criteria misses ~12% of patients with infection and organ dysfunction. It is also important for physicians to recognize two large groups of patients who may not present with the classic SIRS before developing severe sepsis and septic shock: the elderly and the immunocompromised.

**TABLE 159.1 DEFINITIONS FROM THE 1991 ACCP/SCCM CONSENSUS CONFERENCE**

|  |   |
|--|---|
| <b>SIRS</b>  | At least two of the following:<br>■ Temperature > 38°C (100.4°F) or < 36°C (96.8°F)<br>■ White blood cell count >12,000/μL or < 4,000/μL OR<br>Bandemia > 10%<br>■ Respiratory rate > 20 breaths/min OR PaCO <sub>2</sub> < 32 mm Hg<br>■ Heart rate > 90 beats/min |
| <b>Sepsis</b>  | SIRS + documented source of infection   |
| <b>Severe Sepsis</b>                                   | Sepsis + organ dysfunction, hypoperfusion, or hypotension   |
| <b>Septic Shock</b>                                    | Severe sepsis + refractory hypotension (SBP < 90 mm Hg despite 30 mL/kg IVF bolus)  |
| SBP, systolic blood pressure; IVF, intravenous fluids. |   |

The immunosenescence of aging increases the risk of infection in the elderly and also blunts the expression of inflammatory cytokines that are partially responsible for the immune response to infection. A greater proportion of elderly patients older than 65 years of age are afebrile despite serious infection. Hypoxia and tachycardia occur less often in the septic elderly population compared to their younger counterparts, while the less-specific signs of tachypnea and altered mental status are more common. Finally, comorbid illnesses and medications can obscure the diagnosis of sepsis (e.g., beta-blocker suppression of tachycardia or medication-induced leukopenia masking leukocytosis).

The category of immunocompromised patients is broad and heterogeneous, including patients with hematologic malignancies, postchemotherapy patients, patients with low CD4 counts secondary to human immunodeficiency virus, patients maintained on immunosuppressant therapy after transplant or for autoimmune illness, and patients with an acquired or inherited immunodeficiency. The degree of immune suppression is varied not only between disease processes but also between patients with the same diagnosis. These patients may present with a single symptom to alert the emergency provider of infection and impending decompensation, often only a single fever. Mild presentations without localizing signs or symptoms quickly progress to severe sepsis and septic shock in the immunosuppressed, most notably in cancer patients with neutropenic fever or posttransplant patients. Relying on the presence of SIRS or culture positivity before administering antibiotics and fluids in these patients could be disastrous.

With the limitations of the SIRS criteria, the 2001 International Sepsis Definitions Conference expanded the list of diagnostic criteria for sepsis but did not change the overall definitions. These additional diagnostic criteria were adapted by the Surviving Sepsis Campaign in 2012 and are listed in [Table 159.2](#). In 2014, SCCM and the European Society of Intensive Care Medicine convened a task force to update the definitions and clinical criteria for sepsis and septic shock. The recommendations of this task force were recently published and include the elimination of the use of SIRS criteria, implementation of a new retrospectively derived Quick Sequential Organ Failure Assessment score, and deletion of the term *severe sepsis*. These recommendations have generated significant controversy, and, at present, it is unclear if they will be widely adopted by clinicians.

**TABLE 159.2 EXPANDED DIAGNOSTIC CRITERIA FOR SEPSIS**

**Sepsis = documented/suspected infection + some of the following:**

**General Variables**

|   |   |
|---|---|
| Fever                                       | Temperature > 38.3°C (101°F)                  |
| Hypothermia                                 | Temperature < 36°C (96.8°F)                   |
| Heart rate                                  | > 90 beats/min or > 2 SD above normal for age |
| Respiratory rate                            | > 20 breaths/min                              |
| Hyperglycemia                               | > 140 mg/dL without diabetes                  |
| Significant edema or positive fluid balance | > 20 mL/kg over 24 hours                      |
| Altered mental status                       |   |

**Inflammatory Markers**

|                     |                       |
|---------------------|-----------------------|
| Leukocytosis        | WBC > 12,000/ $\mu$ L |
| Leukopenia          | WBC < 4,000/ $\mu$ L  |
| Left shift/bandemia | > 10% bands           |
| C-reactive protein  | > 2 SD above normal   |
| Procalcitonin       | > 2 SD above normal   |

**Hemodynamic Variables**

- Arterial hypotension
- BP < 90 mm Hg
  - MAP < 70 mm Hg
  - Decrease in SBP > 40 mm Hg
  - SBP < 2 SD below normal for age

**Markers of Organ Dysfunction**

|                     |  |
|---------------------|--|
| Arterial hypoxemia  | $\text{PaO}_2/\text{FiO}_2 < 300$                                    |
| Acute oliguria      | UOP < 0.5 mL/kg/h for 2 hours (despite adequate fluid resuscitation) |
| Creatinine increase | > 0.5 mg/dL or 44.2 $\mu$ mol/L                                      |
| Coagulation INR     | > 1.5 or aPTT > 60 s abnormalities                                   |
| Thrombocytopenia    | Platelets < 100,000/ $\mu$ L   |
| Hyperbilirubinemia  | Total bilirubin > 4 mg/dL or 70 $\mu$ mol/L                          |

**Markers of Hypoperfusion**

|  |            |
|--|------------|
| Hyperlactatemia                        | > 1 mmol/L |
| Decreased capillary refill or mottling |            |

WBC, white blood count; SD, standard deviation; MAP, mean arterial pressure; UOP, urine output; INR, international normalized ratio; aPTT, activated prothrombin time.

While the debate over the new definitions and criteria continues, it is important to recognize that the SIRS criteria are neither sensitive enough nor specific for sepsis. The presence of only one or two SIRS criteria, especially in the presence of immunosuppression, should prompt physicians to consider sepsis as the etiology for that patient's presentation. The early administration of appropriate antibiotics and fluid resuscitation should not wait until the patient arrives in the intensive care unit. These lifesaving measures should be given as soon as the diagnosis is suspected in order to decrease mortality and

improve outcomes.

## KEY POINTS

- Limiting the diagnosis of SIRS to two or more criteria misses ~12% of patients with infection and organ dysfunction.
- Elderly patients are often afebrile despite serious infection.
- Comorbid illness or medications may obscure the diagnosis of sepsis by blunting SIRS criteria.
- Immunosuppressed patients can rapidly progress to septic shock without initially meeting SIRS criteria.
- New definitions of sepsis and septic shock have recently been published and focus on scoring systems that identify patients with high mortality.

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# PREVENT CATHETER-ASSOCIATED URINARY TRACT INFECTIONS (CAUTIs) IN THE EMERGENCY DEPARTMENT

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Urinary catheters are often inserted in the emergency department (ED), particularly in patients admitted to the hospital. The Healthcare Infection Control Practices Advisory Committee of the Centers for Disease Control and Prevention (CDC) has defined appropriate indications for indwelling urinary catheters ([Table 160.1](#)). Catheters should not be used to obtain urine for testing if a patient is able to void on his or her own. Likewise, an indwelling urinary catheter should never replace attentive nursing care in patients who are incontinent or bedbound.

**TABLE 160.1 APPROPRIATE INDICATIONS FOR INSERTION OF AN INDWELLING URINARY CATHETER**

- Acute urinary retention or bladder outlet obstruction
- Critical illness requiring accurate measurement of urinary output
- Perioperative care for select surgical procedures
- Prolonged patient immobilization (e.g., pelvic fracture, unstable vertebral fracture)
- Promotion of healing of sacral or perineal wounds in incontinent patients
- Comfort care at the end of life

Urinary tract infections are one the most common health care–associated



infections, of which more than two-thirds are attributable to a urinary catheter. In the United States, catheter-associated urinary tract infection (CAUTI) rates range from 1.3 to 5.3 per 1,000 urinary catheter days in adult critical care areas and 0.2 to 3.3 per 1,000 urinary catheter days on adult inpatient wards. Most CAUTIs are associated with limited morbidity. For some patients, however, a CAUTI can progress to life-threatening bloodstream infection with a mortality rate as high as 15%. Duration of urinary catheterization is the key risk factor for developing CAUTI. Women, the elderly, and those with diabetes mellitus are also more prone to CAUTI. Lapses in sterile technique during urinary catheter insertion and failure to maintain a closed drainage system likewise increase the risk of infection.

Emergency physicians can prevent CAUTI through avoidance of unnecessary urinary catheter insertion. A deliberate assessment of whether an appropriate indication exists for inserting an indwelling urinary catheter should be made. In a cross-sectional study of EDs using administrative discharge data, 8.5% of admitted ED patients received a urinary catheter, of which nearly two-thirds were deemed potentially avoidable. Patients with congestive heart failure who receive a diuretic may not require a urinary catheter to accurately measure urinary output if they can void on their own and reliably collect the urine. A similar case can be made for other stable and alert patients admitted to lower-acuity inpatient wards requiring urine output monitoring. Intermittent catheterization in select patient populations, including those with spinal cord injury or bladder emptying dysfunction, may also present a safer alternative to an indwelling urinary catheter.

Strict aseptic technique should be used when placing an indwelling urinary catheter. This should be preceded by, and concluded with, good hand hygiene (i.e., alcohol hand rub or soap and water). Sterile gloves, a drape, and other necessary supplies should be used. The urethral meatus should be cleansed with an antiseptic solution. Sterile single-use lubricant should be applied to facilitate the passage of the smallest acceptable catheter to minimize urethral trauma. Once the catheter has been secured, the collecting bag should be kept below the level of the bladder but off the floor to promote the unobstructed flow of urine. Kinks in the catheter and tubing should be avoided. Any deviation in aseptic technique or compromise of the closed drainage system resulting in disconnection or leakage should prompt the replacement of the entire catheter and collecting system.

While not all indwelling urinary catheters are avoidable during the initial phase of patient care in the ED, limiting the duration of catheterization can mitigate the risk of CAUTI. A urinary catheter should be promptly removed once it is no longer indicated and as the patient's clinical status improves. Through appropriate and judicious use of urinary catheters, emergency

physicians can play a vital part in preventing CAUTI and optimizing the safety of patients admitted to the hospital.

## KEY POINTS

- CAUTI can progress to bloodstream infection with significant mortality.
- Risk factors for CAUTI include female sex, advanced age, and diabetes mellitus.
- The majority of catheters placed in the ED are potentially avoidable.
- Insert indwelling urinary catheters based on appropriate clinical indications and not solely for convenience.
- If a urinary catheter is necessary, the risk of CAUTI can be reduced by strictly adhering to aseptic technique during insertion, properly maintaining the collecting system, and removing the catheter at the earliest opportunity once it is no longer required for patient care.

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# KNOW THE EMBOLIC COMPLICATIONS OF INFECTIVE ENDOCARDITIS

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Infective endocarditis (IE) is a bacterial or fungal infection of the heart valves or perivalvular structures and is a highly morbid condition associated with significant in-hospital mortality. The morbidity of IE is mediated through direct effects on the heart and embolic complications. Direct cardiac effects of IE include valve incompetence, perivalvular abscesses, conduction abnormalities, and heart failure. Noncardiac sequelae of IE result from embolization of vegetation fragments to select organs. Timely diagnosis of IE and IE-related cardiac dysfunction is imperative to prevent increased morbidity and mortality.

The presentation of IE can be subtle. Often, patients will present with symptoms attributable to multiple organ systems. The majority of patients will have a fever upon presentation. The presence of a new heart murmur has been shown to be a sensitive finding in patients with IE. Classic signs of IE such as Osler nodes, Janeway lesions, and Roth spots are infrequently seen. The Duke Criteria ([Table 161.1](#)) can be used for timely diagnosis and risk stratification of patients with IE.

**TABLE 161.1 MODIFIED DUKE CRITERIA FOR INFECTIVE  
ENDOCARDITIS**

**Major Criteria**

1. Blood cultures positive
  - (a) Typical pathogens derived from 2 separate blood cultures (*S. viridans*, *S. bovis*, *S. aureus*, HACEK) in the absence of a primary focus
  - (b) Persistently positive blood cultures
  - (c) Single positive blood culture for *Coxiella burnetii* or antiphase I IgG titer > 1:800
2. Evidence of endocardial involvement
3. Positive echocardiogram for IE
  - (a) Oscillating intracardiac mass on valve or supporting structures
  - (b) Abscess
  - (c) Dehiscence of prosthetic valve
  - (d) New valvular regurgitation

**Minor Criteria**

1. Predisposing heart condition or IVDA
2. Temperature > 38°C
3. Vascular phenomena, major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, Janeway lesions
4. Immunologic phenomena: glomerulonephritis, Osler nodes, Roth spots, rheumatoid factor
5. Microbiologic evidence: positive blood culture but does not meet a major criterion as noted above or serologic evidence of active infection with organism consistent with IE

**Definitive IE**

1. Histopathologic confirmation OR
2. 2 major criteria OR
3. 1 major, 3 minor OR
4. 5 minor

**Possible IE**

1. 1 major and 1 minor OR
2. 3 minor

**Rejected IE**

1. Firm alternate diagnoses explaining clinical evidence for endocarditis
2. Resolution of IE syndrome with ≤ 4 days of antibiotics
3. No histopathologic evidence of IE on surgical pathology or autopsy after ≤ 4 days of antibiotics
4. Does not meet criteria as above.

Septic embolization occurs in many patients with IE and is due to fragmentation of a valvular vegetation. Risk factors for embolization include diabetes, age, vegetation length, atrial fibrillation, and IE due to *Staphylococcus aureus*. The predominant organs affected by septic embolization depend on the affected heart valve. IE that involves the mitral or aortic valve more frequently embolizes to the brain, spleen, liver, kidneys, and musculoskeletal system. IE that affects the tricuspid valve usually embolizes to the lungs. Right to left shunts, such as those found with a patent foramen ovale, can result in paradoxical septic embolization.

Tricuspid valve IE is typically found in intravenous drug abusers (IVDA), patients with indwelling venous catheters, or patients with automated implantable cardiac defibrillators or pacers. Characteristically,

patients with tricuspid valve IE have a fever and respiratory symptoms. A chest x-ray (CXR) typically demonstrates multiple areas of opacities. Computed tomography (CT) is more sensitive than CXR in diagnosing pulmonary septic emboli. These are often seen as numerous peripheral nodules on CT.

Mitral or aortic valve IE is more predominant in the general population than is tricuspid valve IE. Central nervous system (CNS) embolization can occur in up to 20% and often precedes the diagnosis of IE. Risk of cerebral embolism is higher with mitral valve involvement and *S. aureus* IE. The clinical manifestations depend on the extent and location of the embolic lesions. The territory supplied by the middle cerebral artery is the most common location for embolic lesions. CNS embolization can manifest as an ischemic stroke, hemorrhagic stroke, transient ischemic attack, or simply asymptomatic lesions found on imaging. Less common clinical manifestations include embolic meningitis and mycotic aneurysms. Diagnosis of acute neurologic changes must occur within the context of the presentation. Acute neurologic symptoms in the setting of a fever and heart murmur should raise suspicion for cerebrovascular embolization. The risk of CNS embolization decreases once antibiotics are administered.

Involvement of the coronary arteries is a potentially deadly sequela of IE. Perivalvular abscess formation can develop and lead to extrinsic compression of the coronary arteries. Mitral valve vegetations can embolize to the coronary arteries and lead to an acute coronary syndrome. This, too, generally occurs in a clinical scenario that suggests IE. In the setting of ECG changes consistent with a STEMI, urgent revascularization is still required.

The presence of hepatic or splenic abscesses found on imaging should raise suspicion for IE. Embolization to the kidneys can also lead to abscess formation and bacteriuria. The presence of *S. aureus* in a patient without indwelling catheters and without recent instrumentation should raise suspicion for IE.

It is important for providers to be familiar with the prevalence of IE in their population. Lesions suspicious for embolic phenomenon can be an important clue in making an early diagnosis of IE and avoidance of further complications.

## KEY POINTS

- Timely diagnosis of IE can reduce morbidity and mortality.
- IE can cause significant organ dysfunction through embolization of

vegetation fragments.

- Tricuspid valve IE often causes septic pulmonary emboli.
- Mitral or aortic valve IE can embolize to the CNS, liver, spleen, or kidneys.
- Consider IE in the patient with acute CNS symptoms, fever, and a new heart murmur.

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## THE DON'T MISS DIAGNOSIS: ACUTE RETROVIRAL SYNDROME

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MD

Acute retroviral syndrome (ARS), also called acute human immunodeficiency virus (HIV) or antiviral syndrome, is a clinical manifestation of hyperinfection with HIV. ARS occurs in 40% to 90% of patients with new HIV infection. Unfortunately, the diagnosis is missed in up to 75% of patients. It is characterized by nonspecific complaints that can be attributed to any number of other infections or illnesses. Signs and symptoms typically occur 1 to 4 weeks following exposure and can last from a few days to several weeks. ARS has no racial or gender predilection and is reported in individuals of all ages. Certain modes of transmission are thought to lead to higher rates of ARS manifestations. Suspicion for the disease, recognition of the clinical manifestations, and appropriate testing of high-risk patients can lead to early detection, notification, and treatment. This, in turn, can save lives and greatly limit the spread of HIV.

The most common symptoms of ARS include fever, chills, fatigue, swollen glands, sore throat, and muscle aches. In rare cases, ARS can present as organ failure, stroke-like syndromes with cranial or peripheral nerve palsies, anemia, thrombocytopenia, or the unmasking of malignancy or other indolent viral infections such as Cytomegalovirus, Epstein-Barr, herpes, varicella, or hepatitis. Additional clinical manifestations of ARS are listed in [Table 162.1](#).

**TABLE 162.1 MOST COMMON SYMPTOMS AND FINDINGS IN ACUTE**

## RETROVIRAL SYNDROME<sup>A</sup>

- Fever
- Fatigue
- Rash
- Headache
- Myalgia
- Pharyngitis
- Lymphadenopathy
- Arthralgia
- Night sweats
- Aseptic meningitis
- Gastrointestinal disturbance

<sup>a</sup> In order of decreasing prevalence.

Since ARS is seen in patients with new infection, common HIV antibody tests (i.e., ELISA and Western blot) will be negative. The provider should send serum for HIV ribonucleic acid (RNA). A positive HIV RNA, defined as >50,000 copies, can diagnose ARS. HIV RNA can be detected in as little as 10 days after acute infection. At this time, the virus can replicate to as high as a few million copies in <5 days. After the acute syndromic phase has passed, HIV RNA may decrease to undetectable levels. Standard antibody HIV tests may then become positive. During the period when HIV RNA is undetectable and HIV antibodies are yet to turn positive, CD4 counts can be normal to low normal. It is vital to recognize that patients are highly infectious during this time. With this being said, ~30% of patients with HIV are asymptomatic, which poses an epidemiologic challenge at controlling this global pandemic.

Treatment of ARS remains controversial. The best practice in many of the leading infectious disease literature and consortiums remains unclear and is likely determined in consultation with an experienced infectious disease clinician or virologist. Early treatment can lead to relief of symptoms as well as halt the rapid viral replication characteristic of this phase of infection. Early treatment has also been shown to decrease the decline of CD4 counts



over time with continued and consistent use of antiretroviral therapy (ART). The public health benefit of early initiation of ART is well documented, with a significant reduction of HIV transmission among virally suppressed individuals.

## KEY POINTS

- The diagnosis of ARS is missed in up to 75% of patients.
- Suspect ARS in patients who present with nonspecific symptoms and have risk factors for HIV.
- Antibody testing will not be positive in ARS. If clinically suspected, antigen testing for HIV RNA is appropriate.
- For individuals diagnosed with ARS, early therapy may improve symptoms, preserve CD4 counts, and reduce transmission.
- If positive diagnosis is made, refer to state laws for partner notification services.

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## WEB SITE

[www.hivguidelines.org](http://www.hivguidelines.org)

# **UNDERSTAND WHEN AND HOW TO INITIATE HIV POSTEXPOSURE PROPHYLAXIS IN THE EMERGENCY DEPARTMENT**

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Although a definitive course remains elusive, antiretroviral therapy (ART) has revolutionized the treatment of human immunodeficiency virus (HIV) infection. HIV has the potential to infect health care providers through occupational exposures. More commonly, individuals can become infected with HIV through unprotected sex and intravenous drug use. While exposure avoidance remains the best way to prevent infection, timely postexposure prophylaxis (PEP) utilizing short-term ART reduces the risk of infection after a significant exposure. Emergency physicians should be able to recognize high-risk occupational and nonoccupational exposures, initiate PEP based on current guidelines, and understand the importance of appropriate postexposure follow-up and testing.

## **OCCUPATIONAL POSTEXPOSURE PROPHYLAXIS**

Occupational PEP refers to ART for health care personnel (HCP) who have been exposed to blood, tissue, or other body fluids that can transmit HIV (e.g., cerebrospinal, synovial, pleural, pericardial, peritoneal, or amniotic fluid; semen or vaginal secretions). Saliva, nasal secretions, sputum, sweat, tears, urine, emesis, and feces are unlikely to harbor HIV unless visibly

bloody. Occupational exposures encompass percutaneous injury (e.g., needlestick) or direct inoculation of mucous membranes or nonintact skin (e.g., body fluid splash). The risk of HIV transmission after a percutaneous needlestick is ~0.3%. Transmission is more likely with a deep injury, injury with a device that is visibly contaminated with a source patient's blood, injury with a needle placed directly into a source patient's vein or artery, injury with a hollow-bore needle, or injury involving a source patient with advanced HIV or high viremia. In contrast, the risk of HIV transmission after a mucous membrane or nonintact skin exposure is <0.09%. Intact skin is effective protection against HIV infection; therefore, contamination of intact skin with blood or other body fluids is not considered a significant exposure.

Initial care of the HCP should consist of washing exposed skin with soap and water, irrigating exposed mucous membranes with water or saline, and cleansing small wounds or punctures with an antiseptic. An occupational health or infectious disease specialist should be notified immediately to help assess whether a high-risk exposure warranting PEP has occurred. If the HIV status of the source patient is unknown, a rapid HIV test should be performed, along with testing for other blood-borne pathogens (i.e., hepatitis B and C). If HIV testing cannot be done expediently and the exposure is deemed to be high risk, PEP should be initiated without further delay. If a known HIV-infected source patient has an undetectable viral load, PEP should still be offered as the risk of HIV transmission cannot be entirely eliminated even with effective viral suppression.

## **NONOCCUPATIONAL PROPHYLAXIS**

## **POSTEXPOSURE**

Nonoccupational PEP is prescribed to patients exposed to HIV in the context of unprotected sex, sexual assault, or intravenous drug use. The risk of HIV transmission is greatest with receptive anal intercourse (138 infections per 10,000 exposures) followed by insertive anal intercourse (11 infections per 10,000 exposures), receptive penile-vaginal intercourse (8 infections per 10,000 exposures), and insertive penile-vaginal intercourse (4 infections per 10,000 exposures). The risk of HIV transmission with receptive oral or insertive oral sex is low, but not zero. Factors that increase the likelihood of transmission include if the source patient has acute retroviral syndrome, advanced HIV, or high viremia or the uninfected individual has genital ulcer disease. The risk of HIV transmission in needle-sharing injection drug use is 63 infections per 10,000 exposures to an infected source.

Guidelines for nonoccupational PEP continue to evolve. When the source

patient is known to be HIV infected, the Centers for Disease Control and Prevention recommends PEP if the exposure occurred within 72 hours of presentation and involved a body fluid known to transmit HIV at a body site capable of serving as a point of entry for HIV. The decision to initiate nonoccupational PEP hinges upon the cost of ART, the likelihood of patient adherence to taking ART, and the potential for drug toxicities. PEP is not recommended for patients who repeatedly engage in high-risk behaviors. PEP should be offered to sexual assault victims despite limited data, particularly if the assailant cannot be rapidly tested or has risk factors for HIV infection. Additionally, sexual assault involving anal or genital trauma or multiple assailants may also increase the risk of HIV acquisition.

## **POSTEXPOSURE PROPHYLAXIS REGIMENS AND FOLLOW-UP**

Once the decision has been made to proceed with PEP, administer ART immediately, preferably within the first hours of exposure. PEP is less likely to be effective after 72 hours. Occupational PEP guidelines issued by the U.S. Public Health Service recommend a regimen consisting of three or more antiretroviral drugs. This has been translated to nonoccupational PEP as well. Currently accepted regimens combine a dual nonnucleoside/tide reverse transcriptase inhibitor backbone with either an integrase inhibitor or a protease inhibitor for a total of 28 days. Emergency physicians should consult the most recent PEP guidelines to confirm appropriate first-line and alternative regimens. In addition, infectious disease specialists and the National Clinician's Post Exposure Prophylaxis Hotline (PEP Line) (1-888-448-4911) can serve as invaluable resources in ED PEP-related decision-making.

Patients receiving PEP should have baseline complete blood cell count with differential and renal and hepatic function testing performed during the initial ED evaluation as well as at 2 and 4 weeks following initiation of PEP to evaluate for drug toxicity. Patients should undergo baseline and follow-up HIV testing to evaluate for seroconversion; most seroconversions occur within the first 3 months of exposure. Referral to and coordination of postexposure care by occupational health for work-related exposures or an infectious disease specialist for all other exposures is critical to optimize patient outcomes and provide appropriate counseling and follow-up testing. In some cases, it may be advisable to provide the patient with only a 3- to 7-day supply of PEP to encourage active engagement of these specialists for continuation of care.

## KEY POINTS

- Saliva, respiratory secretions, sweat, tears, urine, emesis, and feces are unlikely to transmit HIV unless grossly bloody.
- High-risk occupational exposures are primarily percutaneous injuries involving deep injury, visible bloody contamination or recent presence of the device in a source patient's vein or artery, hollow-bore needle injury, or injury involving a source patient with advanced HIV infection.
- High-risk nonoccupational exposures include receptive and insertive anal intercourse, receptive and insertive penile-vaginal intercourse, and intravenous drug use.
- PEP is most likely to be effective within 72 hours of the exposure.
- Patients require close follow-up to monitor for PEP drug toxicities, provide appropriate counseling, and repeat testing for HIV and other potential pathogens.

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# **RECOGNIZE THE PRESENTATION OF BIOTERRORISM AGENTS**

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While the majority of providers will never have to act with regard to a bioterrorism incident, rapid recognition of these agents and prompt response is critical to limit morbidity and mortality. The Centers for Disease Control and Prevention (CDC) has broken down bioterrorism agents into three categories (class A, class B, class C) based on ease of spread and potential for significant mortality. This chapter will focus on the class A agents: anthrax, botulism, plague, poxviruses, tularemia, and the viral hemorrhagic fevers. The most important tenets to emergency department management are early recognition, notification of public health authorities, and activation of disaster protocols if there is potential for widespread disease.

## **BACILLUS ANTHRACIS (ANTHRAX)**

There are three major forms of anthrax: cutaneous, gastrointestinal (GI), and inhalational. GI and inhalational anthrax are the biggest concern for bioterrorism, as they have mortality rates of ~50% once symptoms appear. Symptoms of GI anthrax include nausea, vomiting, hemorrhage, and sepsis, whereas inhalational anthrax produces dyspnea, cough, and a hemorrhagic mediastinitis. Though anthrax is not spread from human to human, respirators and protective clothing are recommended until decontamination occurs in the affected area. Treatment is with ciprofloxacin or doxycycline.

## **CLOSTRIDIUM BOTULINUM TOXIN (BOTULISM)**

The bacterium *C. Botulinum* creates a toxin that causes neuromuscular blockade via presynaptic acetylcholine release. Entrance into the body is most commonly by the GI tract or open wounds, though weaponized versions result in inhalation of the bacteria. Botulism is characterized by bulbar nerve palsies and a descending paralysis. Nausea and vomiting can be seen if absorbed via the GI tract. Respiratory failure can occur and is the most common cause of death in botulism. Botulism is diagnosed via assay from serum, emesis, and stool or by bacterial growth on specific medium. The primary treatment of botulism is antitoxin. All-cause mortality ranges from 1% to 17%. Only standard isolation precautions are required for most cases of botulism. Droplet precautions should be used when there is concern for inhalational botulism.

## **YERSINIA PESTIS (PLAGUE)**

There are three major types of plague: bubonic, septicemic, and pneumonic. The majority of natural cases in the United States are bubonic. Bubonic plague causes significant lymphadenopathy, especially in cervical or axillary areas. The pneumonic form can rapidly progress to septicemia and is the most lethal form of plague. Mortality is high even if antibiotics are promptly started. Symptoms of pneumonic plague include fever, malaise, and cough that quickly progress to cardiorespiratory collapse. Doxycycline and ciprofloxacin are the treatment of choice. Simple standard isolation and protection is required for the bubonic form, whereas droplet precautions are required for suspected pneumonic plague.

## **VARIOLA MAJOR (SMALLPOX)**

Though smallpox was eradicated in 1980, samples remain in two level 4 biosafety facilities in the United States and Russia. The virus is spread through droplets, aerosol, and contaminated fomites. Symptoms are initially nonspecific and include fever, generalized pain, and headache. Skin findings initially develop as vesicles and then progress to pustules. In contrast to chickenpox, the lesions of smallpox are all at the same stage of healing and appear centrifugally. The CDC recommends strict airborne and contact isolation for suspected smallpox patients. Mortality from smallpox ranges between 30% and 90% and is dependent upon the pathogenic pattern.

## **FRANCISELLA TULARENSIS (TULAREMIA)**

Tularemia is a naturally occurring infection that can be weaponized. It is

highly infective and easily aerosolized. It is generally spread through contact with infected tissue or arthropods. The ulceroglandular form of tularemia is the most common and produces fever, local ulcers, chills, and headache. Aerosolized versions cause typhoidal tularemia, which is characterized by fever, cough, and chest pain. Treatment of tularemia is streptomycin, gentamicin, or doxycycline. Standard isolation precautions are the current recommendations for contact with patients suspected of harboring the disease.

## **VIRAL HEMORRHAGIC FEVERS**

Viral hemorrhagic fevers are a group of RNA viruses that cover a wide geographic distribution and taxonomic variation. The most common symptoms of these agents are malaise and fever. They can be distinguished from typical viral illnesses by their proclivity to cause significant vascular damage. This leads to significant hemorrhage, multiorgan failure, and shock. Droplet precautions are required, with special attention to bodily fluids. Because this is a large group of uncommon pathogens, consultation with local and state health departments is recommended for assistance with diagnosis. Morbidity and mortality vary significantly and depend on the virus and health care setting. Supportive care is the mainstay of treatment, though ribavirin may play a role in the prevention of viral shedding.

Reporting and surveillance testing varies by local, district, and state health departments. It is important to notify the proper authorities as soon as a potential biologic agent is suspected to ensure timely dissemination of information to health care workers and governmental agencies, thereby resulting in rapid source identification and outbreak containment.

### **KEY POINTS**

- Respirators and protective gear are recommended for anthrax until decontamination of aerosolized spores occurs.
- The pneumonic form of plague can rapidly progress to septicemia and shock.
- Botulism is characterized by bulbar nerve palsies and respiratory failure.
- The lesions of smallpox are classically all in the same stage of healing and spread centrifugally.
- Public health authorities should be urgently notified and appropriate disaster protocols activated if a class A agent is suspected.



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# STAPHYLOCOCCAL TOXIC SHOCK SYNDROME: Do Not Hesitate— Resuscitate

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In the late 1970s, a new critical illness affecting young, otherwise healthy females emerged and was characterized by fever, hypotension, diffuse rash, and multiorgan injury. The U.S. Centers for Disease Control and Prevention (CDC) launched a national surveillance program to elucidate the cause of this highly morbid condition. The surveillance program found an association with menstruation and the use of high-absorbency tampons, with especially high rates in patients who used one particular brand. This brand was subsequently removed from the market, and a more specific link was made to an exotoxin-producing strain of *Staphylococcus aureus*.

Although tampons were the first to be linked to staphylococcal toxic shock syndrome (TSS), there are many other causes. Contraceptive sponges and diaphragms, nasal packing, postpartum infection, mastitis, osteomyelitis, surgical wounds, undrained abscesses, and ulcers have all been linked with staphylococcal TSS. There are also occasional cases in which no cause is identified.

The clinical manifestations of staphylococcal TSS are caused by cytokine-mediated responses to the TSST-1 toxin. These responses lead to the clinical symptoms of fever, malaise, vomiting, diarrhea, myalgias, and dizziness. These nonspecific symptoms can easily be dismissed as symptoms of influenza or other viral syndromes, especially early in the syndrome. The presence of hypotension and altered mental status will raise concern for a more serious infection. Mucous membrane hyperemia (conjunctivae,

oropharynx, genitalia) and a diffuse erythematous rash should alert the clinician to suspect staphylococcal TSS. This characteristic rash is generalized, reddened, flat, and nonpruritic. Desquamation of the hands (dorsal and palmar surfaces) and soles of the feet occurs between 3 days and 2 weeks after onset. While hand and foot desquamation makes the diagnosis of TSS more obvious, it should not be discounted if these signs are absent. Many patients with staphylococcal TSS will present prior to the onset of desquamation.

The CDC criteria for staphylococcal TSS require the following clinical features:

- Fever: temperature  $\geq 38.9^{\circ}\text{C}$  or  $104.0^{\circ}\text{F}$
- Rash: diffuse macular erythroderma
- Desquamation: 1 to 2 weeks after onset of rash
- Hypotension: systolic blood pressure  $\leq 90$  mm Hg for adults or  $<5$ th percentile by age for children  $<16$  years of age
- Multisystem involvement: three or more of the following organ systems
  - Gastrointestinal: vomiting or diarrhea at onset of symptoms
  - Muscular: severe myalgia or creatine phosphokinase level  $\geq 2\times$  upper limit of normal
  - Mucous membranes: conjunctival, oropharyngeal, or vaginal hyperemia
  - Renal: blood urea nitrogen or creatinine  $\geq 2\times$  upper limit of normal or urinary sediment with pyuria
  - Hepatic: total bilirubin, alanine aminotransferase enzyme, or aspartate aminotransferase enzyme  $\geq 2\times$  upper limit of normal
  - Hematologic: platelets  $<100,000/\text{mm}^3$
  - Central nervous system: altered mental status without focal findings in absence of fever or hypotension

Aggressive treatment with intravenous fluids, broad-spectrum antibiotics, and source control should be instituted as soon as possible. Even if the clinical suspicion for staphylococcal TSS is high, the initial emergency department antimicrobial management should remain broad until other causes can be excluded. TSS caused by group A *Streptococcus* (GAS) presents very similarly (termed Streptococcal TSS) but usually has a very obvious source and rarely presents with the rash. Rocky Mountain spotted fever, rubella, and leptospirosis can also present with signs and symptoms similar to those of staphylococcal TSS.

Labs and imaging should be directed at culturing potential sources and monitoring for complications. Staphylococcal TSS can cause disseminated

intravascular coagulopathy, acute respiratory distress syndrome, and myocardial failure. Patients should be admitted to a monitored setting, usually an intensive care unit. When source control is more complicated than tampon removal or abscess drainage, early surgical consultation may be necessary.

The most critical step in making the diagnosis of staphylococcal TSS is to simply consider the diagnosis. If symptoms are suspicious for TSS, search for a source and remove it as soon as possible. It may make the difference between life and death for these patients.

## KEY POINTS

- Suspect staphylococcal TSS in patients presenting with septic shock, diffuse macular erythroderma, and mucosal involvement.
- Desquamation of the hands and feet occurs days to weeks after onset of illness. Do not exclude TSS if patients lack this exam finding at the time they present to the emergency department.
- Always perform a thorough genitourinary exam if suspecting TSS.
- Staphylococcal TSS has also been associated with contraceptive sponges and diaphragms, postpartum state, operative incisions, nasal packings, iatrogenic foreign bodies, abscesses, and chronic wounds.
- Treat with broad-spectrum empiric antibiotics, source control, and supportive critical care.

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# DO NOT BE MISLED BY THE TRADITIONAL MYTHS OF DIARRHEA

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Acute diarrhea is a common complaint of adults and children alike in the emergency department (ED). Safe antidiarrheal therapies exist for all patients with acute diarrhea, and very few patients should leave the ED without symptomatic treatment.

A common myth is that antimotility agents increase the length of disease in infectious diarrhea and are dangerous to patients. The literature for this myth is rooted in a single paper from 1973 in which 25 patients with shigellosis-induced bloody diarrhea were randomized to various treatments including diphenoxylate hydrochloride with atropine (Lomotil). The patients treated with Lomotil had a longer duration of fever and were less likely to clear the infection. The body of literature supporting loperamide with simethicone in watery, nonbloody diarrhea is much more robust and includes multiple randomized controlled trials and systematic reviews that show significant benefit. These trials, however, generally excluded patients with a temperature above 39°C, patients with bloody diarrhea, and hospitalized patients. Current recommendations encourage the use of loperamide with simethicone for adult patients with acute watery diarrhea. While there are data suggesting it may be safe in children over the age of 3, current recommendations are to avoid using loperamide in children for fear of precipitating hemolytic-uremic syndrome (HUS) caused by Shiga toxin from *E. coli* O157:H7. Retrospective studies have shown that antimotility agent

use in children is a risk factor for developing this disease.

For adult outpatients with nonbloody diarrhea, loperamide with simethicone is proven to be a safe and effective treatment and should be prescribed. For providers outside of the United States, the antisecretory agent racecadotril is a well-tolerated alternative.

In patients in whom loperamide is considered unsafe, empiric antibiotics may be an appropriate choice. There is evidence that patients with “traveler’s diarrhea” benefit from a short course of antibiotics if their symptoms are moderate to severe. Treatment is usually with a fluoroquinolone or trimethoprim-sulfamethoxazole (TMP/SMX) for 3 to 5 days. Patients with bloody diarrhea or high fevers are more likely to have an invasive bacterial etiology, which may derive greater benefit from antibiotics. Elderly patients and immunocompromised patients may also benefit from antibiotic therapy. Antimotility agents are traditionally avoided in patients with suspected *Clostridium difficile* infections, though there is little evidence to say they are truly harmful. However, these patients are more likely to improve with antibiotics targeted toward *C. difficile* infection. With antibiotic treatment, there are always potential harms, such as increasing resistance patterns, rash, yeast infections, and tendon rupture. Providers must weigh the risks and benefits of prescribing antibiotics for each patient. In pediatric patients with *E. coli* O157:H7 infectious diarrhea, there is one small study that demonstrates an increased risk of developing HUS when patients were given either TMP/SMX or a cephalosporin. Therefore, routine antibiotics should generally be avoided in the pediatric population or in any patient with high clinical suspicion of enterohemorrhagic *E. coli* (EHEC).

In patients in whom both loperamide and antibiotics are contraindicated, there are other options for symptomatic control of diarrhea. Bismuth subsalicylate (Pepto-Bismol) has been shown to be more effective than placebo in control of acute diarrhea. While not as effective as loperamide, the same safety concerns in terms of prolonging disease are not applicable (though patients should be cautioned about black discoloration of their tongue and stools). While bismuth subsalicylate is contraindicated in pediatrics due to the salicylate, there is still a safe, alternative therapy to help with symptomatic control. Probiotics have been shown to be more effective than placebo in pediatric patients with acute diarrhea, and there are no concerns for disease prolongation or precipitation of HUS.

Do not be misled by the traditional myth of infectious diarrhea. Antimotility agents are effective and safe treatment options for most patients to speed the resolution of diarrhea. For adult patients with nonbloody stools, loperamide with simethicone is most effective. Patients with bloody stools or

high fever will likely benefit from select empiric antibiotics. Lastly, probiotics are a safe choice for symptomatic relief in pediatric patients.

## KEY POINTS

- The majority of patients discharged from the ED with a diagnosis of acute diarrhea should be provided with symptomatic control.
- Loperamide plus simethicone is the most effective choice for controlling nonbloody diarrhea in afebrile adult patients who have a low likelihood of EHEC or *C. difficile* infection.
- Empiric antibiotics are likely indicated in adult patients with bloody diarrhea or high fevers, as they are more likely to have an invasive bacterial etiology, with an important exception for patients with suspected EHEC infection.
- Bismuth subsalicylate improves diarrhea and is safe in most adult patients when both loperamide and antibiotics are contraindicated.
- Probiotics are a great choice for helping to relieve diarrhea in children.

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# **MENINGITIS DOESN'T HAVE TO BE A PAIN IN THE NECK!**

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Meningitis can be caused by infectious or noninfectious etiologies. Noninfectious etiologies of meningitis include malignancy, autoimmune conditions, medications, trauma, and iatrogenic procedures. Infectious meningitis can be caused by a variety of bacterial, viral, fungal, and parasitic organisms. Bacterial and viral organisms account for the most common types of infectious meningitis. In general, viral meningitis is a self-limited disease that resolves with supportive care. In contrast, bacterial meningitis is associated with significant morbidity and mortality. This chapter will focus on pearls and pitfalls in the diagnosis and treatment of patients with bacterial meningitis.

## **PHYSICAL EXAMINATION**

It is commonly taught that patients with bacterial meningitis will exhibit nuchal rigidity, Kernig sign, or Brudzinski sign. Unfortunately, these classic physical examination findings are of limited value in the evaluation of patients with suspected meningitis. In fact, the sensitivity of Kernig's, Brudzinski's, and nuchal rigidity for meningitis is very low; these signs are present in <33% of patients with meningitis. Furthermore, large studies have reported that the classic triad of fever, neck stiffness, and altered mental status is present in just 44% to 66% of patients with meningitis. A comprehensive review evaluated the utility of the physical examination in the diagnosis of patients with confirmed meningitis and found that no individual clinical finding had significant sensitivity or specificity to exclude meningitis. In this study, the highest pooled sensitivities were for headache



(0.50, CI: 0.32 to 0.68) and nausea with vomiting (0.30, CI, 0.22 to 0.38). In contrast to nuchal rigidity, Kernig sign, and Brudzinski sign, jolt accentuation (headache with horizontal rotation of the head at 2 rotations per second) has been shown to increase the probability of meningitis in patients with a fever and headache. In fact, the sensitivity of jolt accentuation in the diagnosis of meningitis has been found to be 100%, with a specificity of 54%.

## **LUMBAR PUNCTURE**

Given the limitations of the physical examination, a lumbar puncture (LP) should be performed to confirm the diagnosis of meningitis. If possible, the LP should be performed as soon as possible in order to maximize the diagnostic yield of culture of the cerebrospinal fluid (CSF). The CSF can be sterilized within hours of initiating antibiotic therapy. Importantly, antibiotic administration should not be delayed to perform the LP.

## **CEREBROSPINAL FLUID ANALYSIS**

An elevated opening pressure, cloudy CSF color, CSF pleocytosis (i.e., elevated white blood cell count), elevated CSF protein, low CSF glucose, and a CSF glucose-to-blood-glucose ratio of  $<0.4$  support the diagnosis of bacterial meningitis. In general, these CSF studies should be considered as methods to confirm the diagnosis of meningitis (positive likelihood ratios  $>10$ ) rather than methods to exclude the diagnosis (negative likelihood ratios  $<0.1$ ).

## **COMPUTED TOMOGRAPHY**

For most patients with meningitis, a computed tomography (CT) of the head is not required prior to the performance of an LP. Concerning patient characteristics and findings that should prompt a CT head prior to LP include immunosuppression, history of central nervous system disease (i.e., mass lesion, stroke, focal infection), focal abnormality on the neurologic examination, or new-onset seizure that occurs within 1 week of presentation.

## **TREATMENT**

In most cases, the clinical presentation and results of CSF analysis are sufficient to guide treatment and disposition. For patients in whom there is clinical ambiguity, it is best to administer antimicrobial therapy while

awaiting the results of CSF culture, as any delay in antibiotics in cases of confirmed meningitis is associated with a significant increase in mortality. Importantly, antibiotic medications are given at higher doses when there is concern for meningitis. For example, 2 g of ceftriaxone or 2 g of cefotaxime should be administered to immunocompetent patients under the age of 50 years to treat meningitis due to *Streptococcal pneumoniae* or *Neisseria meningitides*. Vancomycin should be administered in suspected cases of methicillin-resistant *Staphylococcal aureus*, whereas ampicillin should be administered to patients with meningitis due to suspected *Listeria monocytogenes*.

In addition to antibiotic therapy, the administration of dexamethasone should be considered in cases of bacterial meningitis. Corticosteroids have been shown to reduce hearing loss and neurologic sequelae in patients with bacterial meningitis. Importantly, corticosteroids have not been shown to reduce patient mortality.

## KEY POINTS

- Do not exclude bacterial meningitis based on the absence of nuchal rigidity, Kernig sign, or Brudzinski sign.
- The presence of jolt accentuation increases the likelihood of meningitis in patients with fever and headache.
- Perform an LP to confirm the diagnosis of meningitis.
- Obtain a CT head prior to LP in immunosuppressed patients or those with a history of central nervous system disease, focal neurologic abnormality, or new-onset seizure that occurs within 1 week of presentation.
- Have a low threshold to administer antibiotics in patients with suspected meningitis.

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## **KNOW EMERGING INFECTIONS**

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Acute febrile illnesses are a common reason patients seek emergency department (ED) care. Though the emergency provider is well versed in the management of common conditions, such as an acute viral syndrome, it is critical to consider emerging infectious diseases in the ED patient who presents with an acute illness. The crucial step in the diagnosis of an emerging infectious disease is to obtain an accurate travel and exposure history. In addition, it is also important to inquire about the patient's immunization history. If an emerging infection is suspected based on the results of a travel, exposure, and immunization history, consultation with local health department personnel and infectious disease providers should occur. Worldwide surveillance systems, such as the Centers for Disease Control (CDC), are also beneficial in determining the likelihood of an emerging infectious disease. If an emerging infectious disease is suspected, appropriate isolation and precautions should be taken until confirmatory tests can be completed. This chapter will discuss the clinical presentation and management of select emerging infections including Middle East respiratory syndrome (MERS), chikungunya, Ebola virus disease (EVD), and measles.

### **MIDDLE EAST RESPIRATORY SYNDROME**

MERS is a novel RNA coronavirus. It is similar to the severe acute respiratory syndrome (SARS) virus, although it is less transmissible than SARS. The clinical presentation of MERS can range from asymptomatic to severe pneumonia or renal dysfunction. Critically ill patients with MERS may present with acute respiratory distress syndrome (ARDS). The diagnosis of MERS requires the presence of fever and pneumonia coupled with travel to an endemic area, or contact with someone from an endemic area, within the preceding 14 days. The diagnosis is confirmed by polymerase chain

reaction (PCR). Treatment of the patient with MERS consists primarily of supportive care and management of ARDS. No specific therapies have shown a survival benefit. Though viral transmission is not completely understood, airborne precautions are currently recommended.

## **CHIKUNGUNYA**

Chikungunya is an Alphavirus that is transmitted by mosquitos. Though first identified in southern Africa, the virus is now found in Asia, Oceania, and the Americas. The incubation period is ~3 days after exposure. The clinical presentation is similar to dengue fever and malaria and is characterized by the onset of high fever, polyarthralgias, myalgias, headache, and a rash. The rash is described as a maculopapular rash that is present on the trunk and can extend to the extremities, palms, and soles. Severe infection with multiorgan failure can be seen in children and adults with significant comorbid illnesses. Laboratory abnormalities of this virus include lymphopenia, thrombocytopenia, and a transaminitis. Definitive diagnosis of chikungunya is made with PCR. No specific therapy is available to treat chikungunya. Anti-inflammatory medications can be used to treat arthralgias and myalgias. No specific isolation is required in the ED.

## **EBOLA VIRUS DISEASE**

EVD is a Filoviridae virus that is endemic to Africa and causes a hemorrhagic febrile syndrome. Based on recent data, the clinical presentation of EVD typically begins with high fever and malaise. Approximately 3 to 5 days later, patients develop abdominal pain, vomiting, and diarrhea. It is at this point that patients are considered to be highly infectious and disease transmission can occur through contact with any bodily fluids. EVD has a very high morbidity and mortality that is thought to be primarily due to hypovolemia. Very few patients actually develop clinically significant hemorrhage. Diagnosis of EVD is with PCR. It is important to note that PCR tests can be negative within the first 72 hours after the onset of symptoms. Treatment of EVD consists of fluid resuscitation and electrolyte repletion. Complete barrier and airborne protection are necessary. Point-of-care testing has been used to limit staff exposure in the care of recent patients with EVD.

## **MEASLES**

Measles is a single-stranded, negative sense RNA virus that is highly contagious. Humans are the only known host of this virus. With lapses in

vaccination, cases of measles have once again been diagnosed in the United States. The clinical presentation of measles begins with high fever, cough, coryza, conjunctivitis, and malaise. Koplik spots can be seen in the oropharynx and are considered pathognomonic for measles. The characteristic rash begins on the head ~2 to 4 days after the onset of symptoms and progresses in a caudal direction. For most patients, measles is a benign illness. However, secondary infections and subacute sclerosing panencephalitis can develop. To diagnose measles, both nasal and throat swabs should be sent for PCR. Treatment is supportive with antipyretics, hydration, and treatment of any secondary infections that have developed. In the ED, patients with suspected measles should wear a mask and be placed in airborne isolation. Though patients with measles can be sent home, it is imperative to notify public health officials prior to ED discharge. Patients should be instructed to quarantine themselves at home until symptoms have resolved.

## KEY POINTS

- An accurate travel and exposure history is critical in suspecting an emerging infectious disease.
- In most cases, PCR can be used to diagnose an emerging infectious disease.
- When an emerging infection is suspected, rapidly place patients in appropriate isolation.
- Supportive care is the mainstay of treatment for most emerging infections.
- Regularly review the CDC Web site for information on emerging infections.

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## TB AND SYPHILIS: INFECTIONS YOU CAN'T FORGET ABOUT

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**KAYLA DEWEY, MD AND GHOFRA NE BENGHANEM, MD**

Tuberculosis (TB) and syphilis are infectious diseases that cause a myriad of clinical manifestations. The variability in clinical presentations, coupled with a wide range of prevalence rates, frequently results in the misdiagnosis of these diseases. In recent years, drug resistant forms of both TB and syphilis have been reported. In fact, multidrug-resistant TB (MDR-TB) has emerged as a serious public health threat in many countries.

### TUBERCULOSIS

*Mycobacterium tuberculosis* is transmitted by respiratory droplet nuclei from coughing and sneezing. In most immunocompetent individuals, cell-mediated immunity develops and contains the initial infection. Patients can then go on to develop latent TB and active TB or be cured. Latent TB can persist for years and is simply defined as contained infection without symptoms of active TB. Patient with latent TB have ~ 5% to 10% risk of developing active disease at some point during their lives. This most commonly occurs within the first 2 years following initial infection.

Active TB is defined by the development of signs and symptoms. These symptoms, along with patients at high risk for active TB, are listed in [Table 169.1](#). Importantly, young children are less likely to present with the typical symptoms listed in [Table 169.1](#). Extrapulmonary manifestations of TB can be seen; symptoms are dependent upon the organ system affected by TB. Extrapulmonary involvement of TB can include lymphadenopathy, meningitis, skeletal disease (Pott disease), abdominal symptoms, and genitourinary symptoms. Extrapulmonary symptoms can be seen in 40% to



75% of patients coinfectd with human immunodeficiency virus (HIV). Patients with HIV have approximately a 10% risk of developing active TB during the course of their illness.

**TABLE 169.1 HIGH-RISK PATIENTS AND SIGNS AND SYMPTOMS OF ACTIVE TB**

| <b>HIGH-RISK PATIENTS</b>                      | <b>SIGNS/SYMPTOMS</b>    |
|--|--------------------------|
| Geography: Asia, South/Central America, Africa | Fever                    |
| HIV/AIDS                                       | Fatigue                  |
| Immunocompromised patients                     | Weight loss              |
| Previous isoniazid treatment                   | Night sweats             |
| Homeless                                       | Chronic cough/hemoptysis |
| Prison population                              | Pleuritic chest pain     |

Emergency department (ED) patients with suspected TB should be immediately placed into airborne isolation. A chest x-ray (CXR) should be obtained. Though patients with active TB may have a normal CXR, common abnormalities include hilar lymphadenopathy, cavitary lesions (particularly in the right middle lobe), and pleural effusions. TB testing should also be sent from the ED. The gold standard remains acid-fast bacilli (AFB) sputum tests to confirm active infection. Tuberculin skin testing and interferon gamma release assays (IGRA) can indicate a prior exposure to TB. Recent studies have shown that IGRAs have a better sensitivity and specificity than do traditional tuberculin skin tests to detect latent TB infection.

Patients known to have latent TB who present with signs or symptoms of active infection should be isolated and admitted. Patients without a history of TB but with high clinical suspicion for disease should also be admitted and placed in isolation. In select patients with active infection, or high clinical suspicion of active infection, TB treatment may be initiated in the ED. This is best initiated in consultation with the inpatient team and an infectious diseases specialist.

## SYPHILIS

*Treponema pallidum* is the causative organism of syphilis. The rate of new syphilis infections has been increasing since 2001. This has significant public health implications, as syphilis infection has been shown to increase the rate of HIV transmission. Infection with syphilis typically occurs via direct

contact with an infectious lesion during sexual contact. This is most often due to microtrauma of the mucous membranes that occurs during sexual activity. The lesions of primary syphilis (i.e., chancres) are the most infectious lesions of syphilis.

ED providers should consider primary syphilis in any patient who presents with a genital lesion. The characteristic chancre of primary syphilis is painless, often goes unnoticed by the patients, and usually heals within 2 to 6 weeks without treatment. Untreated, many patients with primary infection will progress to secondary syphilis. Manifestations of secondary syphilis include a maculopapular rash, lymphadenopathy, and condylomata lata. If left untreated, patients develop latent syphilis and, finally, tertiary syphilis. Tertiary syphilis is characterized by end organ damage due to *T. pallidum*. [Table 169.2](#) lists the signs and symptoms of each stage of syphilis.

**TABLE 169.2 STAGES OF SYPHILIS**

| STAGE                        | SIGNS/SYMPTOMS                                |
|------------------------------|---|
| Primary                      | Chancre                                       |
| Secondary                    | Lymphadenopathy                               |
|                              | Maculopapular rash                            |
|                              | Condylomata lata                              |
|                              | Lymphadenopathy                               |
| Latent                       | Fever/weight loss                             |
|                              | None  |
| Tertiary                     | End organ damage                              |
|                              | Cardiovascular                                |
| Neurosyphilis (at any stage) | Meningitis                                    |
|                              | Tabes dorsalis                                |
|                              | Dementia, psychosis                           |
|                              | Ocular syphilis (uveitis/keratitis/retinitis) |

The diagnosis of syphilis is dependent on the stage of disease. Primary and secondary syphilis can be diagnosed based on characteristic physical examination findings. Rapid plasma reagin and venereal disease research laboratory testing can be performed and is especially helpful in identifying individuals who may be in the latent or tertiary stages of disease.

The primary treatment for syphilis is benzathine penicillin. Patients who have a penicillin allergy can receive a tetracycline (i.e., doxycycline) or a macrolide antibiotic medication. Disposition from the ED is dependent on the stage of syphilis. Patients with primary or secondary syphilis can receive

a single dose of penicillin and be discharged with follow-up testing in 6 and 12 months. Patients with suspected tertiary syphilis should be admitted for further evaluation of their end organ dysfunction and prolonged antibiotic treatment. Patients should be informed that any recent sexual partner should be tested and treated.

## KEY POINTS

- TB and syphilis have a myriad of clinical presentations.
- Identifying high-risk patients is important for the diagnosis of TB.
- Early airborne isolation of patients with suspected active TB is critical.
- While neurologic signs and symptoms are commonly associated with tertiary syphilis, they can occur at any stage of the disease.
- Syphilis increases the transmission of HIV; patients should be tested for both infections.

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# AVOIDING COMMON PITFALLS IN INFLUENZA TREATMENT

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Influenza is an infectious disease caused by influenza A and B viruses that results in yearly outbreaks, most notably during the winter months. In general, influenza is a self-limited disease that manifests as respiratory symptoms, myalgias, fatigue, and fever. Influenza can result in significant morbidity and mortality in high-risk populations, namely, the elderly, young children, pregnant women, and patients with chronic medical conditions. Additional high-risk populations are listed in [Table 170.1](#).

**TABLE 170.1 HIGH-RISK POPULATIONS THAT REQUIRE ANTIVIRALS**

|   |   |
|---|---|
| Age <2 y or >65 y old                                 | Hematologic disease (including sickle cell disease)     |
| Pregnant women (or <2 wk postpartum)                  | Morbid obesity (BMI <sup>a</sup> > 40)                  |
| Pulmonary disease (including asthma)                  | Immunosuppression                                       |
| Cardiovascular disease (except isolated hypertension) | American Indians/Alaska natives                         |
| Chronic liver disease                                 | Nursing home or chronic-care facility residents         |
| Chronic renal disease                                 | Neurologic condition impairing management of secretions |
| Metabolic disease (including diabetes mellitus)       |   |

<sup>a</sup> BMI, body mass index.

The Centers for Disease Control and Prevention (CDC) recommend seasonal vaccination against influenza for nearly all patient populations.

When acute influenza infection is diagnosed, or prophylaxis is required, there are two classes of medications that are available for treatment: the neuraminidase inhibitors and the adamantanes. The CDC recommends that patients with suspected or confirmed influenza who require hospitalization or have evidence of prolonged or complicated infection (i.e., pneumonia), or high-risk patients receive a neuraminidase inhibitor. For patients without risk factors for complicated illness who present within 48 hours of symptom onset, the emergency provider must weigh the risks and benefits of therapy with a neuraminidase inhibitor. The adamantanes (e.g., amantadine, rimantadine) are no longer recommended for the treatment of influenza due to limited spectrum of activity and high rates of resistance.

The neuraminidase inhibitors that have activity against most influenza A and B viruses include oseltamivir, zanamivir, and peramivir. Indications, dosing, and side effects of these medications are summarized in [Table 170.2](#). It is important to consider surveillance data and local, state, and national resistance patterns of influenza when selecting a neuraminidase inhibitor. At the present time, over 99% of seasonal influenza virus is sensitive to oseltamivir.

**TABLE 170.2 COMPARISON OF THE NEURAMINIDASE INHIBITORS**

| <b>DRUG</b> | <b>DOSING</b>                      | <b>COMMON SIDE EFFECTS</b> | <b>SPECIAL CONSIDERATIONS</b>                                    |
|-------------|------------------------------------|----------------------------|--|
| Oseltamivir | 75 mg oral, twice daily for 5 d    | Nausea, vomiting           | 1st line for pregnant women.<br>Requires renal dosing adjustment |
| Zanamivir   | 10 mg inhaled, twice daily for 5 d | Bronchospasm               | Avoid in asthmatics or COPD <sup>a</sup> patients                |
| Peramivir   | 600 mg intravenous, one-time dose  | Diarrhea                   | Requires renal dosing adjustment                                 |

<sup>a</sup> COPD, chronic obstructive pulmonary disease.

Oseltamivir (Tamiflu®) is administered orally, is generally well tolerated, and has been shown to decrease the time to symptom improvement by ~16 hours. Though the evidence remains mixed, a 2015 meta-analysis demonstrated a reduction in hospitalizations and influenza complications in patients with laboratory-confirmed influenza who were treated with oseltamivir.

Zanamivir (Relenza®) is administered by inhalation and has been shown to reduce the time to symptom improvement by ~14 hours. In contrast to

oseltamivir, zanamivir has not been shown to reduce the hospitalizations or influenza complications. Zanamivir can cause bronchospasm and should be avoided in patients with asthma or COPD.

Peramivir (Rapivab®) was approved for use in the United States in 2014 and is the newest neuraminidase inhibitor. It is administered as a single intravenous dose of 600 mg. Peramivir was found to be noninferior to oseltamivir in a multinational trial comparing the two medications. Peramivir should be considered in patients unable to receive oseltamivir or zanamivir and in patients in whom there is concern for medication compliance.

Treatment should not be dependent on laboratory confirmation of influenza. Maximal benefit is achieved with early treatment with a neuraminidase inhibitor. Rapid diagnostic tests lack sensitivity for influenza. In fact, a 2012 meta-analysis demonstrated a pooled sensitivity of just 62% for rapid influenza testing. Given the variable effectiveness of the influenza vaccine, treatment should not be withheld, even in vaccinated patients, when there is a high clinical suspicion of influenza. High-risk patients who are discharged from the emergency department with antiviral treatment should have close outpatient follow-up.

## KEY POINTS

- Treat all high-risk and hospitalized patients with confirmed, or suspected, influenza with a neuraminidase inhibitor, regardless of the time of symptom onset.
- Consider the benefits and harms of treatment in low-risk patients presenting within 48 hours of symptoms onset. Do not treat these patients if they present after 48 hours.
- A history of influenza vaccination does not rule out influenza. Treat the patient if there is high clinical suspicion of infection.
- Do not base the decision to treat patients with suspected influenza on laboratory confirmation.
- Consider patient comorbidities and adherence when choosing a neuraminidase inhibitor.

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# APPROPRIATE ANTIBIOTIC CHOICES FOR RESISTANT ORGANISMS

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Antibiotics are prescribed to ~15% of emergency department (ED) patients and are among the most frequently utilized medications in emergency medicine. Often, the emergency provider (EP) must make clinical decisions on antibiotics based on incomplete, or absent, microbiologic data. Notwithstanding, the list of resistant organisms has grown exponentially. In order to improve outcomes and limit the number of resistant organisms, it is imperative for the EP to carefully select an appropriate antibiotic regimen in the treatment of ED patients with select infectious diseases. This chapter will discuss appropriate antibiotic medications for common ED infections including urinary tract infection (UTI), community-acquired pneumonia (CAP), and skin and soft tissue infections.

## URINARY TRACT INFECTIONS

The most common pathogen responsible for a UTI is *Escherichia coli*. Traditionally, trimethoprim-sulfamethoxazole or ciprofloxacin has been used to treat uncomplicated UTIs in ED patients. Importantly, resistant rates to these two antibiotics have risen to more than 20% in select regions. Similarly, aminopenicillins and first- and second-generation cephalosporins also have reduced efficacy in the treatment of UTIs. As a result, nitrofurantoin is now recommended as the first-line antibiotic for an



uncomplicated UTI. Current resistance rates for nitrofurantoin are low. Importantly, nitrofurantoin has reduced efficacy for *Enterococcus*, *Klebsiella*, and *Pseudomonas* organisms.

UTIs caused by extended spectrum beta-lactamase (ESBL)–producing organisms are increasing in health care. Up to 20% of infections caused by Enterobacteriaceae are now due to ESBL-producing organisms. Risk factors for infections due to ESBL organisms include age >65 years, multiple comorbidities, nursing home residence or recent hospitalization, and recurrent UTIs. ESBL-producing organisms have high resistance rates to ceftriaxone, piperacillin-tazobactam, and ciprofloxacin. Carbapenem antibiotics are currently the best option for patients with an ESBL-associated UTI. Importantly, these medications should be used judiciously to avoid the development of resistance to this class of antibiotics as well. Urine cultures should be sent for patients at risk for drug-resistant UTIs.

## PNEUMONIA

CAP is a common infectious disease that results in hospitalization from the ED. Standard diagnostic criteria for CAP includes clinical signs and symptoms of pneumonia (i.e., fever, cough, pleuritic chest pain) and the presence of an infiltrate on chest x-ray. Surprisingly, up to 30% of patients diagnosed with pneumonia in the ED do not meet these standard criteria. In addition, many patients have a viral etiology for their pneumonia and do not need antibiotic therapy.

In the United States, *Streptococcus pneumoniae* is the most common bacterial pathogen in CAP. The primary outpatient treatment of CAP in low-risk adult patients is a macrolide antibiotic (i.e., azithromycin). For outpatients with comorbidities, a fluoroquinolone or the combination of a macrolide and  $\beta$ -lactam antibiotic is recommended. Importantly, up to 30% of severe pneumococcal infections are resistant to these first-line antibiotics. Patients who are younger than 5 or older than 65 years of age are at risk for severe pneumococcal infections. In 2010, a 13-valent pneumococcal vaccine was developed that has dramatically reduced the spread of resistant pneumococcal strains. Nonetheless, patients with severe pneumococcal infection are generally admitted.

A macrolide antibiotic in combination with either a respiratory fluoroquinolone or  $\beta$ -lactam is recommended for ED patients with CAP who require admission. For patients with suspected drug-resistant *S. pneumoniae*, *Pseudomonas* infection, or those who are critically ill, an antipseudomonal  $\beta$ -lactam (i.e., piperacillin-tazobactam) should be administered in addition to a macrolide antibiotic. Patient risk factors for infection with drug-resistant

*Pneumococcus* or *Pseudomonas* include chronic heart or lung disease, diabetes mellitus, asplenia, malignancy, immunosuppression, and recent antibiotic use. Vancomycin should also be added to the antibiotic regimen when infection with methicillin-resistant *Staphylococcus aureus* (MRSA) is suspected. Health care–associated pneumonias occur within 90 days of a hospitalization or nursing home admission and carry a high risk of infection with multidrug-resistant organisms. Therefore, broad-spectrum antibiotic coverage is recommended (e.g., combination therapy with piperacillin-tazobactam and vancomycin), with further adjustments based on microbiologic culture data.

## SKIN AND SOFT TISSUE INFECTIONS

ED visits for skin and soft tissue infections (SSTI) have nearly tripled in the last 15 years. Although community-acquired MRSA is commonly implicated as a causative pathogen in SSTI, beta-hemolytic *Streptococcus* species still account for nearly 80% of cases of cellulitis without abscess. These organisms remain susceptible to penicillins and first-generation cephalosporin antibiotics. Treatment for MRSA infection is generally unnecessary for patients with nonpurulent cellulitis. In addition, many cutaneous abscesses can be sufficiently treated with incision and drainage alone, as antibiotic treatment has not been shown to improve cure rates. Treatment for MRSA infection should be considered in patients with open wounds, previous penetrating injury, intravenous drug use, and immunosuppression. Broad-spectrum antibiotics should be reserved for patients with suspected necrotizing skin infections or those with signs of systemic illness. Although vancomycin is currently the first choice for MRSA-associated SSTI, EPs should avoid giving a single dose of vancomycin prior to ED patient discharge. A single dose of vancomycin does not result in therapeutic blood levels and contributes to the development of resistant organisms. Appropriate outpatient regimens for suspected MRSA SSTI include clindamycin, doxycycline, or the combination of trimethoprim-sulfamethoxazole and cephalexin.

### KEY POINTS

- Local microorganism resistance rates and hospital antibiograms should be reviewed when selecting an antibiotic.
- Nitrofurantoin is the first-line antibiotic for uncomplicated UTI.
- A carbapenem antibiotic should be used as a first-line agent to treat a

UTI in patients at risk for infection due to an ESBL-producing organism.

- Consider the possibility of drug-resistant *Pneumococcus*, *Pseudomonas*, or MRSA infection in critically ill patients with pneumonia.
- Avoid MRSA antibiotics in well-appearing patients with simple cellulitis.

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# KNOW INFECTION PREVENTION

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Patients with potential transmissible infections often present to the emergency department (ED) for evaluation and treatment. Given the high patient volume, combined with the high acuity of ED patients, it can be challenging to maintain effective infection control measures to prevent the spread of disease to health care workers and other patients. This chapter will review routine infection prevention measures, along with prevention measures for both common and rare infectious disease outbreaks.

## ROUTINE INFECTION PREVENTION MEASURES

Two tiers of ED infection prevention exist: standard and transmission-based precautions. Standard precautions apply to all ED patients and are the primary prevention strategy for the spread of infectious agents. Transmission-based precautions apply to patients with known, or suspected, infectious agents whose routes of transmission may not be completely prevented by standard precautions. These transmission-based precautions should be applied in the ED while awaiting confirmation of the select pathogens listed in [Table 172.1](#).

**TABLE 172.1 RECOMMENDATIONS FOR TRANSMISSION-BASED PRECAUTIONS WITH SELECTED CASES**

| TYPE OF PRECAUTION | SELECTED CASES   | SPECIFICATIONS   |
|--------------------|--|--|
| Standard           | ALL patients   | <ul style="list-style-type: none"> <li>■ Hand hygiene before/after patient contact</li> <li>■ Gloves, gown, eye protection when contact with bodily fluids</li> <li>■ Safe disposal and cleaning of equipment/linen</li> <li>■ Cough etiquette</li> </ul>  |
| Contact            | Pathogens implicated to spread via environmental contamination<br>( <i>C. difficile</i> , <i>E. coli</i> O157:H7, RSV, HSV, enterovirus, scabies, impetigo, MRSA, VRE, SARS, smallpox, adenovirus)   | <ul style="list-style-type: none"> <li>■ Wash hands with soap and water</li> <li>■ Private room preferred</li> <li>■ Gloves and gown upon entering room</li> <li>■ Consider gowning patient during transport</li> <li>■ Noncritical items should be dedicated to use for a single patient if possible</li> </ul> |
| Droplet            | Pathogens spread through respiratory or mucous membrane contact with respiratory secretions<br>( <i>N. meningitides</i> , <i>H. influenzae</i> type B, Diphtheria, <i>B. pertussis</i> , pneumonic plague, influenza, rubella, mumps, adenovirus, RSV) | <ul style="list-style-type: none"> <li>■ Private room preferred; cohort if necessary</li> <li>■ Wear a surgical mask when within six feet of the patient</li> <li>■ Mask the patient during transport</li> </ul>   |
| Airborne           | Pathogens that remain infectious over long distances in the air<br>(tuberculosis, varicella, measles, smallpox, SARS)  | <ul style="list-style-type: none"> <li>■ Place the patient in a negative pressure room</li> <li>■ Wear a certified respirator (e.g., N-95 or PAPR).</li> <li>■ Mask the patient during transport</li> </ul>  |
| Complete           | High mortality rate, lack of treatment, and/or incompletely defined transmission modes<br>(hemorrhagic fever, Ebola, Marburg, MERS-CoV)  | <ul style="list-style-type: none"> <li>Follow standard, contact, and airborne precautions</li> <li>Complete provider skin coverage and eye protection required</li> <li>Use a trained observer for all PPE</li> </ul>  |

RSV, respiratory syncytial virus; HSV, herpes simplex virus; MRSA, methicillin-resistant *Staphylococcal aureus*; VRE, vancomycin-resistant enterococcus; SARS, severe acute respiratory syndrome; MERS-CoV, middle east respiratory syndrome Coronavirus.

## INFECTION PREVENTION MEASURES FOR COMMON OUTBREAKS

Some infectious organisms have predictable outbreak patterns that may be seasonal or regional or target a specific patient population. ED infection prevention plans should be implemented for common seasonal infectious outbreaks, such as influenza. There are several important steps to prevent the spread of influenza that can be adapted to other commonly encountered infectious outbreaks. These steps include the following:

- Administration of vaccines to patients and health care personnel. This is the most important measure to prevent seasonal influenza infection.
- Minimize potential exposures by posting visual alerts to ensure all persons with symptoms of a respiratory infection adhere to respiratory hygiene, cough etiquette, and hand hygiene.
- Institute triage screening procedures to rapidly identify possible influenza patients.
- Remove ill health care providers from direct patient care.
- Adhere to standard and droplet precautions for patients with suspected influenza.
- Train health care providers in the transmission of infectious agents, including influenza.

## **INFECTION PREVENTION MEASURES FOR RARE EPIDEMIC OUTBREAKS**

The recent Ebola virus outbreak highlights the importance of having robust organizational infrastructures to prevent the spread of novel infectious agents. EDs should establish clear infection control plans for novel infections that have a high mortality rate, high risk of human-to-human transmission, and lack of available vaccines or effective treatment. EDs should implement stringent triage protocols to rapidly identify potential patients with these conditions. Every patient with a potential infectious disease should be asked about travel history. In addition, emergency providers should be knowledgeable on current epidemiologic patterns of emerging pathogens.

Once a novel pathogen is suspected, the patient should be immediately isolated in a predefined area of the ED. In addition, it is important to limit the number of health care providers who have direct contact with the patient. Health care providers assigned to work with patients under investigation for a novel pathogen should be appropriately trained in proper donning and doffing of personal protective equipment (PPE). Unfortunately, the rate of retention of correct donning and doffing procedures 6 months after training has been shown to be poor. Due to the complexity of the donning and doffing process, a checklist should be developed and followed. In addition, a trained

observer should supervise the donning and doffing procedure to ensure it is properly executed.

It is also critical to use an anteroom in order to clearly separate clean and contaminated areas. Urgent consultation with facility infection control specialists and referral to specialty care should be obtained early in the diagnostic process for these patients. Perhaps most importantly, an onsite manager should oversee the care of patients under investigation for a novel pathogen in order to ensure appropriate infection prevention measures are followed.

## KEY POINTS

- Hand hygiene is the single most important measure to reduce transmission of microorganisms.
- Early screening of all ED patients with infectious symptoms for an epidemiologic link to communicable diseases is vital to prevent the spread of infection.
- Post visual alerts in waiting rooms, and ask patients to adhere to respiratory hygiene, cough etiquette, and hand hygiene.
- Transmission-based precautions should be implemented while awaiting the results of confirmatory tests.
- A trained observer should oversee donning and doffing of PPE in order to prevent the spread of novel infectious agents.

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## TREATING PNEUMONIA IN COPD

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**DIANA LADKANY, MD AND JEFFREY DUBIN, MD, MBA**

Chronic obstructive pulmonary disease (COPD) is a term used to describe several pulmonary diseases, including emphysema, chronic bronchitis, and small airway disease. COPD is characterized by alveolar destruction with a loss of pulmonary elasticity, airway inflammation, hypersecretion of mucous, and fibrosis. Factors predisposing to COPD include chronic infections, environmental exposures (e.g., cigarette smoking), and genetic factors. Complications of COPD regularly encountered in the emergency department include acute exacerbations with bronchospasm or pneumonia. Both conditions can result in long hospital or intensive care unit stays and the need for mechanical ventilation and are associated with increased mortality in COPD patients. In the United States, there are over 1 million hospital admissions and more than 50,000 deaths due to pneumonia annually. Severe pneumonia occurs more frequently in patients who have been hospitalized, receive dialysis, are immunosuppressed, or have chronic cardiac, liver, or pulmonary disease.

COPD is an independent risk factor for mortality in patients with pneumonia. Specifically, there is a higher mortality rate if the patient is intubated or fails a trial of noninvasive mechanical ventilation. Although combined inhaled corticosteroids and long-acting beta agonists reduce the number of acute COPD exacerbations, a recent trial demonstrated an increase in mortality for COPD patients treated with chronic inhaled corticosteroids who developed pneumonia. Fluticasone-salmeterol is associated with a higher mortality rate in COPD patients with pneumonia compared with budesonide-formoterol.

It is important to remember that lung flora is altered in COPD patients, which places them at risk for colonization with resistant organisms. Possible explanations for the alteration in flora include frequent infections and the

chronic use of inhaled corticosteroids. These factors alter lung epithelial immunity and allow bacteria to proliferate within the airway. In addition, numerous viruses can also be found on PCR testing in the setting of acute COPD exacerbations. Furthermore, influenza virus denudes the airway epithelium, can precipitate a viral pneumonia, and increases the risk for superimposed bacterial infections. It is difficult to determine which of these pathogens is clinically significant. Given the alterations in lung architecture, it can be difficult to diagnose pneumonia on the chest x-ray (CXR) of a COPD patient. In fact, lobar consolidation is often not seen in COPD patients with a clinically significant pneumonia.

The type of pneumonia (community-acquired, health care–associated, and hospital-acquired) is categorized by the different pathogens found in sputum samples from the patient. *Streptococcus pneumoniae* remains the most common pathogen in community-acquired pneumonia, but it is not the only important pathogen in COPD patients with pneumonia. The most common pathogens associated with pneumonia in COPD patients include *Haemophilus influenza*, *Moraxella catarrhalis*, *S. pneumoniae*, and *Pseudomonas aeruginosa*. Antibiotic therapy must be tailored to treat these pathogens. It is also important to consider treatment with oseltamivir within the first 48 hours of symptoms during influenza season.

The recommendations for inpatient antibiotic treatment of COPD patients with pneumonia include an antipseudomonal beta-lactam (i.e., piperacillin-tazobactam, cefepime, imipenem, meropenem) and a respiratory fluoroquinolone or an aminoglycoside antibiotic. Outpatient treatment recommendations for COPD patients with pneumonia include combination therapy with a fluoroquinolone and a macrolide. Empiric treatment should consist of combination therapy until sputum culture results and sensitivities can be used to guide therapy.

## KEY POINTS

- COPD is an independent risk factor for pneumonia and mortality.
- Inhaled corticosteroids are effective at decreasing COPD exacerbations but increase the overall mortality in COPD patients with pneumonia.
- The pulmonary epithelium is altered in COPD patients, which results in different pneumonia pathogens.
- The most common pathogens are *S. pneumoniae*, *H. influenza*, *M. catarrhalis*, and *P. aeruginosa*.

- Antipseudomonal antibiotic coverage should be included as part of the antibiotic regimen.

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# DIAGNOSE AND TREAT NECROTIZING SOFT TISSUE INFECTIONS QUICKLY!

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**TABITHA GARGANO, MD AND KORIN HUDSON, MD,  
FACEP, CAQSM**

A necrotizing soft tissue infection (NSTI) is a rare, but serious and rapidly progressive infection of the fascia, subcutaneous tissue, deep dermis, or muscle. NSTIs are characterized by angiothrombotic microbial invasion of the tissues and a liquefactive necrosis. These life-threatening infections have a mortality rate that ranges from 20% to 40%, even when properly treated. Survivors often suffer significant morbidity that may include amputations, extended hospital stays, multiple surgical procedures, and decreased quality of life. The annual incidence of NSTIs is estimated to be between 500 and 1,500 cases and has continually risen over the last decade. NSTIs are notoriously difficult to distinguish from less severe non-NSTIs. The only interventions that have been shown to improve outcome from NSTIs are rapid diagnosis, aggressive resuscitation, and early surgical debridement.

Most NSTIs are polymicrobial and include gram-positive, gram-negative, aerobic, and anaerobic species. The rapid progression and diffuse systemic involvement of NSTIs is largely due to the production of bacterial exotoxins. These exotoxins cause platelet aggregation and microthrombosis, which creates an environment favorable for bacterial replication and also prevents the delivery of antibiotics to the site of infection. In addition to these local effects, exotoxins decrease vascular tone, suppress cardiac function, and cause intravascular hemolysis. Ultimately, this leads to the development of acute kidney injury, acute respiratory distress syndrome, and

A thorough history can help the emergency provider (EP) make the diagnosis of an NSTI. Though up to two-thirds of patients with an NSTI are previously healthy individuals, select comorbid conditions have been shown to be associated with NSTIs. These conditions include diabetes mellitus, advanced age, intravenous drug abuse, immunosuppression, recent surgical procedures, chronic renal or hepatic disease, alcohol abuse, and recent trauma. In addition, the EP should take note of the patient who presents with a soft tissue infection in the setting of current antibiotic use. Current antibiotic use may delay disease progression, alter the course of an NSTI, and cause in a delay in diagnosis.

The clinical presentation of an NSTI can be variable. As a result, many patients are initially misdiagnosed with less severe infections, such as cellulitis, abscess, or erysipelas. Systemic signs such as fever and tachycardia are present in ~50% of patients with an NSTI. Cutaneous findings can include erythema and induration in 65% to 85% of patients. Fluctuance of the area is only found in ~30% of cases. Crepitus, considered a hallmark finding in NSTIs, does not appear until late in the course of disease and is not sensitive for the diagnosis. Additional late examination findings can include bullae, necrosis, and motor and sensory deficits. The most important physical examination findings are pain out of proportion to the skin exam and tenderness that extends beyond the area of erythema. There are no laboratory tests that are sufficiently sensitive or specific to diagnose an NSTI.

When an NSTI is suspected, the Laboratory Risk Indicator for Necrotizing Fasciitis score (LRINEC) may be useful for the EP. This score is listed in [Table 174.1](#). This tool calculates a score based on laboratory values. A score >6 has a positive predictive value of 92% and negative predictive value of 96% for NSTI. While the LRINEC score can be a useful adjunct to a careful clinical evaluation, a low score should not be used to exclude an NSTI when there is a high index of suspicion for the disease.

**TABLE 174.1 LRINEC SCORE**

| LABORATORY VALUE  | POINTS |
|---|--------|
| <b>CRP</b>  |        |
| <150 mg/L   | 0      |
| >150 mg/L   | +4     |
| <b>WBC</b>  |        |
| <15/mm <sup>3</sup>   | 0      |
| 15–25/mm <sup>3</sup>   | +1     |
| >25/mm <sup>3</sup>   | +2     |
| <b>Hemoglobin</b>   |        |
| 13.5 g/dL   | 0      |
| 11–13.5 g/dL  | +1     |
| <11 g/dL  | +2     |
| <b>Creatinine</b>   |        |
| ≤1.6 mg/dL  | 0      |
| >1.6 mg/dL  | +2     |
| <b>Blood Glucose</b>  |        |
| ≤180 mg/dL  | 0      |
| >180 mg/dL  | +1     |
| Adapted from Wong CH, Khin LW, Heng KS, et al. <i>Crit Care Med.</i> 2004;32:1535–1541. |        |

There is a limited role for imaging in the diagnoses of NSTI. Computed tomography with the use of intravenous contrast is the best option, with a reported sensitivity and negative predictive value that approach 100%.

The most important factor in treating NSTIs is early diagnosis. In order to avoid missing this rare, but lethal, condition, EPs must maintain a high index of suspicion and look for signs of systemic involvement. These patients should receive aggressive resuscitation with intravenous fluid therapy and broad-spectrum antibiotics that include a protein synthesis–inhibiting antibiotic (i.e., clindamycin) to reduce toxin production. Early surgical consultation is critical and should occur as soon as the diagnosis is suspected. Emergent surgical debridement remains the treatment of choice for an NSTI.

## KEY POINTS

- An NSTI is a rapidly progressive infection with high morbidity and mortality that is often misdiagnosed.
- Diabetes mellitus, intravenous drug abuse, immunosuppression, recent surgical procedures, and recent trauma are risk factors for NSTI.
- The hallmark findings of an NSTI include pain out of proportion to skin findings and tenderness that extends beyond the area of erythema.
- Treat with aggressive fluid resuscitation, empiric broad-spectrum antibiotics, and early surgical referral.
- Emergent surgical debridement is the treatment of choice for an NSTI.

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# WHAT IS THE BEST WAY TO MEASURE CORE TEMPERATURE?

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**MATTHEW MORRISON, MD**

Temperature is a critical piece of information in the assessment of emergency department (ED) patients. Body temperature is regulated by the hypothalamus and affected by numerous cytokines in the presence of infection. Fever is often the initial manifestation of illness and a critical gauge of thermoregulatory homeostasis. In fact, for many conditions (i.e., appendicitis, neuroleptic malignant syndrome, myxedema coma, heat stroke), an elevated temperature heralds a life-threatening condition. Thus, it is critical to accurately measure temperature in ED patients. This chapter will discuss the methods to measure core temperature, namely, rectal, oral, temporal, and tympanic measurements.

A rectal temperature measurement is considered the gold standard for core temperature assessment and can be performed in ED patients. Numerous studies have demonstrated that a rectal temperature is superior to measurements taken from the oral, temporal, axillary, and tympanic locations. A 2013 study demonstrated that fever was missed in up to 20% of patients when nonrectal locations were used to measure patient temperature. Similarly, a pediatric study demonstrated that fever was missed in up to 40% of febrile children when a tympanic location was used to determine temperature. A common misperception is that 1.0°F (0.6°C) can be added to an oral temperature measurement to approximate the rectal temperature. Various studies, including those performed in intensive care unit and pediatric populations, have failed to consistently confirm the accuracy of this adjustment.

Limitations in the measurement of rectal temperatures include efficiency, patient comfort, and patient privacy. In hypothermic patients, the rectal probe



must be placed 15 cm into the rectum to avoid sampling error. In neutropenic patients, a rectal temperature should not be performed due to the concern of bacterial translocation across the bowel wall with subsequent development of bacteremia.

Oral, tympanic, temporal, and axillary temperatures are imprecise and do not accurately reflect core body temperature. Reasons for this imprecision are multiple and include extremes of ambient temperature, tachypnea, and recent intake of hot or cold beverages. With respect to tympanic measurements, the decubitus position and hair in the external auditory canal have been shown to affect temperature measurements. Temporal measurements are subject to variations in vascular vagaries and adversely affected by perspiration.

It is also important to note that temperature measurements are affected by diurnal variation, gender differences, older age, and immunosuppression. In the event that a rectal temperature cannot be obtained, catheterization of the bladder with a temperature-sensing catheter can be performed to measure core temperature. Bladder temperatures have been shown to be similar to rectal measurements. Finally, an esophageal probe or pulmonary artery catheter can be used to obtain a core temperature, though most EDs do not possess the necessary equipment, or expertise, for these measurements.

## KEY POINTS

- Elevated temperature frequently heralds the onset of life-threatening illness.
- Fever is missed by oral and tympanic methods in up to 20% of patients.
- Do not rely on a correction factor to adjust oral temperatures to reflect rectal measurements.
- Do not perform a rectal temperature measurement in neutropenic patients.
- Rectal temperature is preferred over oral, tympanic, and temporal temperature measurements.

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## **SECTION XII**

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# **MS NONTRAUMA**

## **UGH! ANOTHER REPEAT VISIT FOR BACK PAIN?! KEEP EPIDURAL ABSCCESS ON THE DIFFERENTIAL!**

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**ELAINE HUA SITU-LACASSE, MD**

Back pain is a common emergency department (ED) chief complaint, and the workup is not always easy. It is up to the physician to determine if the patient needs NSAIDs and discharge paperwork versus blood work, antibiotics, and an MRI of the total spine. The key to appropriate management is diagnosis. We will focus on the diagnosis of spinal epidural abscesses (SEAs) in this chapter.

SEAs are rare, but missing one can be devastating for you and for the patient. About half of patients with an SEA are discharged and return for a second ED visit because of their initial nonspecific symptoms.

The most important aspect of the initial evaluation is asking the right questions. The following are common risk factors for SEAs: intravenous drug use, recent hospitalizations, diabetes mellitus, end-stage renal disease, cancer, alcoholism, and anything that may cause immunosuppression. Spinal procedures such as injections, lumbar punctures, epidurals, etc. also predispose patients to SEAs. Beware of red herrings such as recent minor trauma or chronic back pain because they may be obscuring a more insidious cause for the back pain. Don't let them trip you up.

Signs and symptoms are crucial. Classic presentation for an SEA would be a febrile male IV drug user with back pain and neurologic deficits; but patients seldom read our textbooks, so this classic triad is rare, seen in <20% of patients—only about one-third of patients even have fever! The goal of early diagnosis of SEA is the prevention of worsening infection and cauda

equine syndrome. A patient's symptoms and their progression are useful: worsening back pain, weakness, incontinence, decreased sensation, and/or paralysis. This is the time to listen to your patient.

On review of patients' charts, many doctors neglect to document their exam accurately. Documentation of a good physical exam, especially a thorough neurological exam, including a digital rectal exam (DRE) to evaluate sphincter tone, is helpful, especially if the patient returns for the same chief complaint. Make sure to introduce yourself to the patient before the DRE. The patient's sensation, motor strength, cerebellar function, and reflexes should be documented. Don't be lazy; avoid writing "nonfocal neurological exam." Sensory exam should also include evaluation for saddle anesthesia.

Thorough visual examination of the back should be done first, looking for areas of redness, increased warmth, or swelling especially if he/she had a recent procedure. The patient will likely have midline back pain, and it can be worse with percussion of their spinous processes. Everything needs to come off the patient, so an examination of the patient's skin can be done, making sure an abscess, cellulitis or decubitus ulcer is not missed. This also includes looking between the fingers and toes, taking off the adult diaper, etc.

The finding of urinary retention in a patient is more sensitive than other findings on the physical exam. If you are worried about overflow urinary incontinence, a bladder scan should be done before and after the patient pees to check for postvoid residual.

Blood work is straightforward: blood cultures  $\times 2$  if patient is febrile with urinary retention, CBC, CMP, and ESR. The WBC may be normal because only 60% of patients have elevated WBC. ESR will likely be elevated in the setting of SEA, and your spinal surgeons will use it to track disease improvement.

As for imaging studies, forget x-rays. The most appropriate imaging study is the MRI. MRI of total spine with IV contrast will (a) identify the SEA, (b) reveal the extent of the abscess, and (c) may help rule out SEA and rule in other diagnoses such as malignancy, transverse myelitis, hematomas or a bulging disc. If your ED or hospital does not have MRI capabilities or the patient cannot have an MRI done due to a noncompatible device, then the next best thing is CT myelography. Usually performed by a radiologist, a CT myelogram is done by injecting contrast dye into the subarachnoid space to identify any areas of cord compression.

This chapter has mainly focused on diagnosis of SEAs and minimally on management thus far. If the suspicion for SEA is high, start antibiotics early,

especially in the setting of sepsis. The offending organism is usually *Staphylococcus aureus*, but the antibiotics should also cover *Escherichia coli* and *Pseudomonas aeruginosa*, so the suggested regiment is IV vancomycin plus cefepime or meropenem. Advocate for the patient and make sure he/she is next on the list for the MRI. The sooner the SEA is diagnosed, the sooner the patient can get to the operating room. Small abscesses may be drained by interventional radiology, but your spinal surgeon should be contacted first. If your hospital does not have a spine surgery service, the patient should be transferred as quickly as possible to a center that has one. Delays in transferring may mean development or worsening of neurologic deficits while in your ED, and you do not want that.

## KEY POINTS

- The classic triad of fever, back pain, and neurologic deficits is uncommon, so a good history and thorough physical exam will assist with making the diagnosis.
- Don't deprive your patient of the "ED handshake"; the physical examination should always include a DRE.
- Normal temperature and WBC are commonly seen in these patients, so they are not dependable to completely rule out an SEA.
- STAT MRI total spine with IV contrast should be ordered as soon as possible.
- If suspicion is high and the patient is septic, IV antibiotics should be started early and spine surgery should be involved ASAP.

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## IF YOU SUSPECT THE HORSE'S TAIL, CHECK THE SADDLE!

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As a clinician in the emergency department, almost every shift will involve taking care of a patient with back pain. Nearly 90% of individuals experience an episode of back pain at some point.<sup>1</sup> Many cases of back pain will resolve in 4 to 6 weeks with minimal intervention and without significant sequela. However, there are multiple causes of back pain that can lead to significant morbidity for the patient and serious medicolegal consequences for the emergency department physician if not promptly diagnosed.<sup>2</sup> By seeking out red flags in the history and physical, the clinician in the emergency department can avoid unnecessary imaging while identifying patients with do-not-miss diagnoses.

Cauda equina syndrome (CES) is a do-not-miss cause of back pain. The cauda equina, named for its resemblance to a horse's tail, is a bundle of nerve roots beginning at the L1 vertebral level. CES is caused by compression of the cauda equina. Although the condition was first described in 1934, no formal definition exists.<sup>2</sup> The causes of CES are numerous. Large, central herniation of an intervertebral disc, frequently at the L4–L5 level, remains the most common cause.<sup>3</sup> Other causes include epidural abscess, neoplasm, spinal hematoma, spinal stenosis, ankylosing spondylitis, and trauma.

It is paramount that the emergency physician has a high index of suspicion to detect CES among the many patients presenting with back pain. Unfortunately, symptoms and signs in early CES may be subtle. Most delays in time from presentation to treatment occur at the time of diagnosis.<sup>4</sup>

Therefore, you must have a systematic approach to avoid potential misdiagnoses.

A systematic history and physical will often contain clues to a more serious underlying etiology. “Red flags” in the history that suggest a bad etiology of back pain include age <20 or >50 years, duration of symptoms (>4 weeks), presence of systemic symptoms (fever, weight loss), history of trauma, history of injection drug use, immunocompromised status, neurologic deficits below the waist, bowel dysfunction, and urinary retention, which may be accompanied by overflow incontinence. Of these, saddle anesthesia, lower extremity weakness, and loss of bowel or bladder function are the classic triad of symptoms associated with CES. Yet, CES may present in a variety of ways. Other symptoms associated with CES include back pain, bilateral sciatica, sexual dysfunction, gait disturbances, and paralysis. One study of CES patients presenting to the emergency department found the most common symptoms to be pain, flaccidity, and difficulty with ambulation.<sup>5</sup> Unfortunately, if gait disturbances or urinary retention are present upon presentation, many patients will continue to have some degree of deficit post treatment.<sup>1</sup>

A thorough and systematic examination is paramount. Red flags that may be revealed during the physical examination include fever, positive straight leg raise test, neurologic deficits including saddle anesthesia, areflexia, or abnormal gate. Physical examination of a patient with back pain should include examination of the spine, including evaluation for dermatologic changes, point tenderness, and deformity. The physical examination should also include strength testing; sensory testing, specifically of the “saddle” region and lower extremities; digital rectal examination; deep tendon reflex testing; and assessment of gate. Do not skip the rectal examination!<sup>2</sup> The digital rectal examination may only be skipped if the patient lacks a rectum. Diminished rectal tone is present in ~60% to 80% of CES cases.<sup>1</sup> Among physical exam findings, saddle anesthesia has a sensitivity of 0.75.<sup>6</sup> So if you suspect the horse’s tail is involved, check the saddle!

In addition to the physical exam, a postvoid residual bladder catheterization must absolutely be performed if CES is suspected. Urinary retention (>100 mL) has a sensitivity of ~0.9 and a negative predictive value of 99.99%.<sup>1</sup> In fact, urinary retention of 500 mL or more combined with bilateral sciatica, subjective complaints of urinary retention, and/or rectal incontinence has been found to be predictive of MRI-confirmed CES.<sup>7</sup>

Laboratory testing is frequently directed at specific causes of CES, such as infection or neoplasm. A complete blood cell count, an erythrocyte



sedimentation rate, and a urinalysis are among the more commonly ordered tests.<sup>1,8</sup> MRI remains the gold standard for diagnosis due to superior soft tissue differentiation.<sup>1,9</sup> If CES is suspected, an MRI should be obtained emergently. If MRI is not available or contraindicated due to metal implants, CT myelography may be used. IV steroids are routinely administered in both traumatic and atraumatic CES, although evidence to support use in atraumatic CES is controversial. Dexamethasone and methylprednisolone are commonly used agents. Prompt surgical consultation is required, as decompression performed within 48 hours is the current stand of care,<sup>2</sup> although timing of surgical intervention also remains controversial.<sup>3</sup>

## KEY POINTS

- CES is rare cause of back pain that can lead to significant morbidity if not promptly diagnosed.
- A systematic history review and physical examination, including digital rectal examination, is critical for early diagnosis of CES.
- Always check a postvoid residual bladder catheterization! Normal urinary retention is (<100 mL).
- MRI is the diagnostic tool of choice.
- If CES is confirmed by MRI, IV steroids should be considered and a prompt surgical consultation obtained.

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# **UNDER PRESSURE: RAPIDLY DIAGNOSING AND TREATING ACUTE COMPARTMENT SYNDROME OF THE EXTREMITIES**

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**ANNA L. WATERBROOK, MD**

Acute compartment syndrome (ACS) is a true life- and limb-threatening surgical emergency, which requires a high degree of clinical suspicion to make the diagnosis. If it is recognized and treated early enough, it may prevent permanent damage to limb tissues, resulting in severe disability including paralysis, contractures or loss of limb, rhabdomyolysis, kidney damage, and possibly death.

ACS occurs anytime there is increased pressure in a musculoskeletal compartment that compresses muscles, nerves, and vessels and results in decreased tissue perfusion, ischemia, and eventually cell death. While it may affect any enclosed tissue space, it most commonly occurs in the muscle compartments of the arms and legs. The lower leg contains four compartments surrounding the tibia and fibula, which are the anterior, lateral, superficial posterior, and deep posterior compartments, and accounts for 40% of ACS. The forearm contains both a volar or flexor compartment and a dorsal or extensor compartment and are the next most commonly affected. Other areas, such as the hands, feet, and thigh, may also be affected, but much less commonly.

While there are many potential causes of ACS, it most often occurs following a fracture. The most common fractures associated with ACS include tibia, humeral shaft, forearm, and supracondylar fractures. Other

causes include prolonged limb compression or immobilization (i.e., comatose, intubated, prolonged surgeries, casts, tourniquets, etc.), excessive exertion, crush injuries, reperfusion injury, burns, intravenous drug use, coagulopathy, and envenomations.

ACS is largely a clinical diagnosis and one needs a high degree of suspicion in order to recognize it. Symptoms usually rapidly progress over a few hours but can be delayed 48 hours after the initial event. The classic teaching for the signs and symptoms of ACS are the “5 P’s” (pain, paresthesias, paresis, pallor, and pulselessness). These however are not very sensitive or specific for ACS and only rarely will all be present. Pain out of proportion to exam is the most sensitive finding in ACS. The patient will often experience pain even with passive stretch. Most commonly, the affected compartment will be swollen, firm, and tender with squeezing by the examiner and is often described as “woody.” Paresthesias may begin within 2 to 4 hours of elevated compartment pressures. Paresis (weakness) may often be subtle and missed because it is attributed to pain. Pallor and pulselessness are late findings. Usually, the arterial circulation is not absent, and thus, the affected extremity will still be warm and have a pulse. When pulselessness does occur, it is usually long after tissue necrosis and ischemia have occurred.

Whenever the diagnosis is in doubt, it is essential to measure compartment pressures. Normal compartments pressure is <10 mm Hg. Classic teaching is that compartment pressures that exceed 30 mm Hg need fasciotomy. However, there are newer studies that advocate using a difference in pressure of <30 mm Hg between intracompartmental pressure and the diastolic blood pressure to determine when fasciotomy is necessary. Studies have shown that using the delta p decreases chances of unnecessary fasciotomies while not leading to any increased morbidity or mortality. This is because compartment pressures necessary for injury vary based upon systemic blood pressure. Hypertensive patients may require much higher elevations for tissue ischemia to occur, while patients who are hypotensive may have tissue damage occur at much lower pressures.

Compartment pressures may be tested with a commercially available device, which includes a manometer, a needle, and a syringe with normal saline. It is important to not only measure the compartment in question but also the surrounding compartments. In order to measure compartment pressures, place the needle in the appropriate compartment, inject a couple of drops of normal saline, and record pressure. It is recommended to check each compartment twice. If there is an associated fracture, it is important to measure the compartment pressure within five centimeters of the fracture site. If a commercially available device is unavailable, it is also possible to

measure compartment pressures using an 18-gauge needle attached to an arterial line setup. It is important to remember that a normal pressure does not rule out ACS. If clinical suspicion is high, perform serial measurements and obtain early surgical consultation. It may also be prudent to consider laboratory evaluation such as creatinine kinase, myoglobin, and urinalysis to evaluate for rhabdomyolysis.

Treatment of ACS is aimed at preventing irreversible tissue damage and its subsequent complications. Early surgical consultation for fasciotomy is essential and the only effective or definitive treatment. Permanent damage is unlikely to occur if ACS is treated within 6 hours of injury; however, irreversible damage results when there has been over 8 hours of tissue ischemia. Initial treatment includes removal of any compressive dressings or casts, analgesics, supplemental oxygen, and a normal saline bolus if the patient is hypotensive to improve tissue perfusion. Keep the affected limb at level of the torso (i.e., the heart), in order to maximize perfusion.

## KEY POINTS

- ACS is a true life- and limb-threatening surgical emergency and can be a difficult diagnosis to make.
- ACS is primarily a clinical diagnosis. Do not rely on compartment pressures if clinical suspicion is high.
- The most sensitive signs and symptoms in ACS are pain out of proportion to exam and a tense compartment.
- Use  $\Delta P$  (diastolic blood pressure-intercompartmental pressure)  $<30$  mm Hg to help determine if fasciotomy is necessary.
- Any delay in diagnosis or treatment can lead to significant morbidity, thus early surgical consultation for fasciotomy in any suspected case is essential.

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compartment syndrome of the leg. *J Orthop Trauma*. 2008;22(8):581–587.

# PHYSICAL EXAM AND BLOODWORK Do Not Adequately Differentiate Infectious From Inflammatory Arthritis

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DERICK D. JONES, MD, MBA AND CASEY M.  
CLEMENTS, MD, PhD

Septic arthritis can irreversibly destroy and damage a joint if not diagnosed and treated rapidly. How does this happen? Bacteria within a joint produce an inflammatory response that chews up cartilage, prevents new cartilage from forming, and can even lead to pressure necrosis of the surrounding joint structures. If this process is not stopped rapidly, outcomes could be functional deterioration of the joint, fusion, or even joint amputation. You don't want to be responsible for any of these outcomes in your patients. If that wasn't bad enough, septic joints are associated with mortality rates of 10% to 15% varying with comorbidities. Complications such as bacteremia, sepsis, endocarditis, and distributive shock are not uncommon. Given the concerns for joint damage and increased mortality, rapid diagnosis and treatment of a septic joint are crucial. Unfortunately, in the ED, if you rely solely on physical exam, history, and bloodwork, you aren't going to be able to reliably differentiate a septic joint from an inflamed joint, and you put yourself and the patient at risk of missing the diagnosis.

Convention would have you believe that a septic joint will present as a warm, red, painful swollen joint with restricted motion, and when you see this, you should tap. In fact, studies have shown that joint pain, history of joint swelling, and fever are the **only** findings that occur in greater than 50%

of patients with a confirmed septic joint. Chills and sweats occur in a minority of patients. This means that we need a much higher index of suspicion for septic joints as many patients do not present with “classic” findings. Combinations of “positive” exam findings increase the likelihood of a septic joint, while absence of these same findings does not substantially reduce the risk of disease in an acutely painful and swollen joint (8% to 27%). Therefore, a clinician cannot rely on their physical exam findings alone to change to prescribe or withhold treatment with confidence. We have to do the right test: tap the joint!

Several risk factors, such as a history of diabetes, rheumatoid arthritis, prior joint surgery, prosthesis, and overlying infection, have been shown to increase the pretest probability of septic arthritis; however, the absence of these does not significantly reduce the probability to rule out joint infection. Exceptions are history of prosthesis with overlying cellulitis (+LR 15) and recent joint surgery (+LR 6.9), which when positive can substantially increase the likelihood of infection and help rule in the disease. Regardless, one would likely tap in these cases for diagnostic confirmation.

Blood tests are not as helpful as you would think. Many studies have investigated the accuracy of leukocytosis for the diagnosis of septic arthritis but have used varying thresholds. Regardless of threshold, no studies have demonstrated an acceptable sensitivity or overall diagnostic accuracy of peripheral WBC count for septic arthritis. Inflammatory markers, like ESR and CRP, have reasonable sensitivity; however, there is no cutoff for ESR or CRP that significantly increases or decreases the posttest probability of septic arthritis. Bacterial response markers such as procalcitonin have the same problem. In summary, blood tests provide interesting information but are useless in ruling in or ruling out the disease and should not alter your management.

Given the poor predictive value of physical exam and serologic studies, if septic arthritis is suspected, you should tap the joint and send the fluid for culture, Gram stain, leukocyte count with differential, and crystal evaluation. Fluid culture will be positive in most patients with bacterial arthritis but has high false-negative rates and should not be used alone to rule out the disease. In addition, they may be falsely negative if the patient has received recent antibiotics. Synovial white blood cell count can significantly increase or reduce the posttest probability of septic arthritis depending on synovial fluid leukocyte count. Studies have shown that values of  $0$  to  $25 \times 10^9/L$  significantly reduce the posttest probability of septic arthritis and values greater than  $50 \times 10^9/L$  significantly increase the posttest probability of septic arthritis. Intermediate values require careful consideration. Some have



advocated empiric antibiotics with intermediate-risk testing results, though this is controversial.

To manage joint infections, blood cultures should be drawn, and orthopedic surgery should be consulted early for surgical management in addition to antibiotic therapy. All patients should be admitted to the hospital for continued treatment. Tap the joint if you are considering infection, avoid these diagnostic pitfalls, and sleep better at night (or during the day).

## KEY POINTS

- Septic arthritis can lead to irreversible joint damage and increased morbidity and mortality. A low index of suspicion for testing should be maintained in the ED population, and early consultation with orthopedics is essential.
- Physical exam and history should not be relied upon for the diagnosis of septic joint. Their absence does not rule out disease.
- Blood tests in general are not useful acutely.
- Inflammatory markers such as ESR and CRP do not significantly increase or decrease the posttest probability of disease at any cutoff level.
- Arthrocentesis is the definitive diagnostic test.

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# **DON'T GET BROKEN UP ABOUT MUSCLE BREAKDOWN**

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Rhabdomyolysis is a clinical condition due to muscle necrosis and the release of intracellular contents into circulation. Manifestations of this condition can be broad, ranging from asymptomatic to critical illness, with the most concerning complications being hyperkalemia, renal failure, and, rarely, disseminated intravascular coagulation.

Etiologies of rhabdomyolysis can be divided into four categories: impaired production or use of ATP, dysfunctional oxygen or nutrient delivery, increased metabolic demand exceeding capacity, and direct myocyte damage. Rhabdomyolysis should be considered in patients with polytrauma or crush injuries, restrained combative patients, immobilized patients, and electrical or burn injuries. Rhabdomyolysis can also occur in overexertion of normal muscle groups and should be considered in scenarios of heat exhaustion, heat stroke, as well as temperature dysregulation scenarios such as malignant hyperthermia or neuroleptic malignant syndrome. Increased sweating can cause hypokalemia, and the resulting potassium depletion can impair muscle perfusion. Impaired oxygen delivery, as also seen in sickle cell or high altitude, results in muscle ischemia and can lead to rhabdomyolysis. Seizures, psychosis, or drug-induced agitation create hyperkinetic states that can result in muscle breakdown. Infection and inherited disorders are more commonly seen etiologies in children.

The classic triad of symptoms includes muscle pain, weakness, and dark urine, though not all three symptoms may be present in pediatric populations. Presenting physical exam findings vary depending on severity but can include muscle tenderness, edema, or weakness with vital sign abnormalities including tachycardia and fever. Physical exam should include careful

evaluation of the skin for crush injuries, bruising, and compartment abnormalities. Presenting symptoms can be vague, so detailed history taking is key in the proper identification of rhabdomyolysis, noting alcohol or drug use, heat exposure, or trauma. Medication history should take careful note of antipsychotics and statins, the most common prescription-offending agents.

When initiating your emergency department workup, laboratory studies will be your guiding diagnostic tools. Findings include elevated creatine kinase (CK) levels, electrolyte abnormalities, metabolic acidosis, myoglobinuria, and acute kidney injury, as seen in more severe cases. CK levels begin to rise 2 to 12 hours after onset of injury. Diagnosis of rhabdomyolysis should be considered when the CK level is at or above five times the upper limit of normal at presentation, ~1,000 IU/L. Decline in levels begins at 1 to 3 days, and re-evaluation of continued injury should be performed if levels do not decline appropriately.

Emergency department management includes identification and correction of the underlying etiology, aggressive fluid resuscitation, and correction of metabolic and electrolyte abnormalities as needed. While not universally accepted, some advocate for urinary alkalization for renal protection. Extremities should be evaluated carefully for the development of compartment syndrome, with appropriate monitoring and intervention as needed. Severe cases of renal failure and electrolyte and metabolic abnormalities may require dialysis for correction.

Hypovolemia occurs secondary to third spacing and contributes to further renal injury, making fluid resuscitation a critical intervention. Potential electrolyte abnormalities include hyperkalemia, hyperphosphatemia, hypocalcemia, and hyperuricemia. Each results from damage to the muscle cells and release of intracellular contents. Monitoring urinary output is important as oliguria is an ominous sign. AKI is a common complication, with increased incidence in cases associated with dehydration, sepsis, and acidosis. AKI is more common with CK levels of >5,000 IU/L. A rare complication, disseminated intravascular coagulation, can be seen as a result of the release of thromboplastin and prothrombotic agents from damaged muscle cells. Most patients with rhabdomyolysis should be admitted to the hospital for continued fluid resuscitation and close monitoring of renal function and electrolytes.

## KEY POINTS

- Clinical manifestations of rhabdomyolysis include muscle tenderness,

weakness, and dark urine.

- Diagnosis is confirmed by elevation in serum CK levels. Monitoring of serum levels will show a peak in 1 to 2 days with decline after 3 days, if inciting etiology has been corrected.
- Rhabdomyolysis should be considered in a variety of clinical histories including traumatic or crush injuries, drug abuse, seizures, and heat exposure.
- Initial ED management includes the identification and correction of offending etiology, electrolyte management, and fluid resuscitation to prevent more severe sequelae of renal injuries.

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## WHEN BACK PAIN IS AN EMERGENCY

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**JAMES BOHAN, MD**

Back pain is an extremely common complaint in every emergency department (ED). It is easy to become complacent because of how common it is. A great majority of the time, the diagnosis is a nonspecific back pain that is treated with conservative management. As emergency physicians, we need to be vigilant to the emergent causes of back pain, causes that can result in permanent neurologic dysfunction, or even death. Cauda equina syndrome, epidural abscess, malignancy, and ruptured abdominal aortic aneurysm (AAA) are causes we need to watch for and diagnose on evaluation.

Cauda equina syndrome is a rare but serious entity to diagnose and treat. The lumbar spine is composed of 5 vertebrae (L1–L5) followed by the sacrum. Under each vertebral body is the neural foramen that the nerve root passes through with the corresponding number. The spinal cord terminates at the conus medullaris at the level of L1–L2. From that point, the nerve roots are known as the cauda equina. Cauda equina syndrome is most commonly a result of a massive central disc herniation that compresses multiple nerve roots. Other causes include malignancy, trauma, and epidural abscess. The most common symptoms are lower back pain radiating down both legs, saddle anesthesia, and urinary or bowel dysfunction resulting in urinary retention and decreased rectal tone. It is a surgical emergency requiring decompression.

So how does a savvy ER doctor diagnose this? The patient will, of course, present with lower back pain, typically with radicular symptoms, often bilateral. A key finding is urinary incontinence. This is overflow incontinence from a neurogenic bladder. Checking postvoid residual with ultrasound or bladder scanner will confirm this finding (>200 mL).<sup>1</sup> The

patient may also have decreased anal sphincter tone or saddle anesthesia. Saddle anesthesia is sensory deficit in the perineum, buttocks, and proximal thigh (areas you would sit on a saddle). With any of these findings, an emergent MRI is required to make the diagnosis. Outcome is improved if decompression occurs within 2 days of symptom onset.

Epidural abscess is a dangerous spinal infection difficult to diagnose. At higher risk for epidural abscess are IV drug users, immunosuppressed, liver/kidney disease, those who underwent a spinal procedure, diabetics, and alcoholics. Bacteria reach the epidural space hematogenously from a remote site, from a spinal procedure, or local extension from infections such as a disc space infection or psoas infection. *Staphylococcus aureus* is the most common bacterial cause, causing 63% of cases.

One-third of the time, no portal of entry is found. However, the most common portals of entry are following a spinal procedure or from skin and soft tissue infections (especially abscesses).<sup>2</sup> The classic presentation triad is fever, back pain, and neurologic deficit, but to have all three is rare. Often, patients will present without fever and often have no neurologic deficits. Yes, they may just have back pain. No wonder it's a diagnosis often missed on first presentation. The patient will complain of back pain, even at rest, and subjective fevers. There may be tenderness to percussion in the area affected. Helpful laboratory studies are an ESR and CBC, but often, there is no leukocytosis initially. Ultimately, the diagnosis is made by obtaining an emergent MRI with and without contrast. Epidural abscesses are treated with neurosurgical drainage and decompression, in addition to broad-spectrum IV antibiotics. It is important to include vancomycin, given the increased prevalence of MRSA.<sup>3</sup> Blood cultures should be obtained once the diagnosis is made. Lumbar puncture is often not done as the yield is low. In IV drug users, it is important to cover for *Pseudomonas*.

Malignancy in the spine will present with back pain as well. Lesions on the spine can resultant in cord compression, which needs to be emergently treated. Pain is the most common presenting complaint. Classically, it is pain that worsens at night, insidious onset, and partially relieved by activity. A sudden pain may be the result of a pathologic fracture. CT will show the bony detail best; however, MRI is indicated if there are signs of cord compression. MRI with contrast will show the extent of epidural disease, edema around any fracture, and small lesions that may be missed on CT. Treatment includes high-dose steroids and neurosurgical consultation.

Finally, it is extremely important to keep ruptured AAA in your differential of back pain. AAA does not cause symptoms unless it is rapidly expanding or ruptures. A thorough review of AAA is beyond the scope of

this chapter, but is an important differential to consider. Ruptured AAA may present as only back or flank pain with no abdominal complaints. This can often be misdiagnosed as renal colic. Bedside ultrasound or noncontrast CT scan can be used to make the diagnosis.

Cauda equina syndrome, epidural abscess, spinal malignancy, and AAA are “red flag” causes of back pain that need to be in the differential of every patient with this chief complaint. History and exam findings can often eliminate these diagnoses as a cause, but on occasion, imaging is required.

## KEY POINTS

- Urinary retention >200 mL, saddle anesthesia, and back pain are signs to consider cauda equina syndrome and MRI.
- Epidural abscess is often very difficult to diagnose. History of back pain, fever, and neurologic deficits is the classic triad, but often not present. High-risk populations include IV drug users, recent spinal procedure, diabetics, and immunocompromised.
- Spinal malignancy will present with insidious onset of back pain worse at night. CT scan shows structural integrity of bones best, whereas MRI will show edema and small malignancies better.
- Ruptured AAA may present only with flank pain that can be misdiagnosed as renal colic.

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# RHEUMATOID ARTHRITIS AND SPONDYLOARTHROPATHIES

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## RHEUMATOID ARTHRITIS

Rheumatoid arthritis (RA) is a chronic inflammatory disorder affecting predominately joints and ligaments, and occasionally the lungs, arteries, and eyes. Joint involvement is primarily symmetrical involving small joints (hands, wrists), less commonly the larger joints. Two potential errors are worthy of emphasis when evaluating patients with RA in the ED.

*Avoid attributing joint pain, swelling to an exacerbation of the underlying RA, thus not investigating the possibility of infection.* Preexisting joint disease increases the risk of septic arthritis from an incidence of 2 to 5 per 100,000 in the general population to 28 to 38 per 100,000 in patients with RA. Because the prevalence of RA is about 1%, an exacerbation of joint pain and inflammation is more likely rheumatoid than septic. Risk factors for septic arthritis in RA: age > 80 years, injection drug use, superficial skin ulceration or infection, oral carriage of *Staphylococcus aureus*, prosthetic hips or knees, diabetes mellitus, and biologic agent therapy. Two useful clues indicating septic joint in an RA flare are (1) symptoms or signs in one joint out of proportion to others (more pain, swelling, redness, decreased movement) and (2) primarily larger joint involvement. RA flares have predilection for small joints of the hands and feet. Larger joint involvement, without the smaller joints, is uncommon. Other clues to septic arthritis in RA are significant impairment in movement, marked redness and warmth overlying the joint, and occurrence of fever, chills, and sweats.

Joint aspiration for analysis is important. Arthrocentesis is easiest in the knee, where about half of septic arthritis complicating RA occurs. Aspiration



of fluid from other joints (shoulder, elbow, wrist, ankle) is more difficult but may be facilitated by fluoroscopic or ultrasound guidance. Small joint aspiration (MCP, PIP, MTP) is difficult for even experienced technicians.

Delay initiating antibiotic therapy increases the incidence of cartilage and bone destruction producing permanent damage. Highly virulent organisms (*S. aureus*) can produce damage within 4 days, with less virulent bacteria (streptococci) taking up to 10 days. If ED arthrocentesis is unsuccessful, delay up to 12 hours until a subsequent procedure attempt (under guidance?) will not substantially impact outcome. Conversely, initiation of empiric antibiotic therapy before joint aspiration may reduce causal organism identification when fluid is ultimately obtained. Thus, the emergency physician should make a good faith effort at joint aspiration, and if unsuccessful, not initiate intravenous antibiotic therapy, but arrange for the patient to be hospitalized for later arthrocentesis.

*Avoid ascribing neck pain to muscle sprain and not considering rheumatoid involvement of the cervical spine.* Cervical spine involvement with RA is common, radiographically evident in 25% of RA patients under age 40 and up to 85% in those over 60. The three most common sites of involvement are atlantoaxial instability, subaxial instability, and basilar invagination. Manifestations are pain, features due to compression of brainstem or spinal cord (myelopathy), and compression of a cervical nerve (radiculopathy) ([Table 182.1](#)).

**TABLE 182.1 CERVICAL RADICULOPATHY TABLE**

| <b>CERVICAL NERVE ROOT</b> | <b>DERMATOMAL PAIN</b>                  | <b>MOTOR IMPAIRMENT</b> | <b>REFLEX IMPAIRMENT</b> |
|----------------------------|---|-------------------------|--------------------------|
| C2                         | Posterior occiput, temporal             | None                    | None                     |
| C3                         | Occiput, retrobulbar, retroauricular    | None                    | None                     |
| C4                         | Base of neck                            | None                    | None                     |
| C5                         | Lateral arm                             | Deltoid                 | Biceps                   |
| C6                         | Radial forearm, thumb, and index finger | Biceps, wrist extension | Brachioradialis          |
| C7                         | Middle finger                           | Triceps, wrist flexion  | Triceps                  |
| C8                         | Ring and little finger                  | Finger flexion          | None                     |
| T1                         | Ulnar forearm                           | Hand intrinsics         | None                     |

Neck pain is so common; it may be difficult to distinguish rheumatoid complications. Pain from cervical subluxation is typically worsened by movement, especially forward flexion where some describe the sensation that

their head is falling forward. Pain associated with myelopathy or radiculopathy is more specific for significant cervical spine disease but is not specific for RA.

Myelopathy symptoms include manual dexterity impairment or clumsiness with balance and gait difficulties. Myelopathy is associated with atlantoaxial or subaxial instability, typically exacerbated by forward flexion. Differentiating myelopathy features from an underlying disability (joint inflammation, damage, and age) is not easy, and delay in diagnosis is common.

Basilar invagination describes the inflammatory destruction of the C1 vertebra and adjacent joints allowing the skull to settle downward. The odontoid may advance upward, pressing on the medulla. Stimulation of the vomiting center, compression of the respiratory center, and impairment of cranial nerves 9 through 12 are possible.

The majority of RA patients with acute nontraumatic neck pain do not require imaging. However, if neck pain is associated with features of myelopathy (such as acute gait disturbance) or radiculopathy, imaging is indicated. Cervical spine imaging is also indicated when occipital headache is present without neck pain and when new-onset neck pain is present in a patient with long-standing RA with evidence of severe joint damage. Plain films (especially combined with flexion-extension views) have some utility in assessing spinal stability and basilar invagination. However, the ability of MRI to evaluate soft tissues and changes in the brainstem and spinal cord make it the imaging modality of choice.

## **SPONDYLOARTHROPATHIES**

Spondyloarthropathies are a family of chronic inflammatory conditions affecting the axial spine (spondylitis), the sacroiliac joints, and tendon or ligament insertions. Four disorders are considered within this family: ankylosing spondylitis, psoriatic arthritis, reactive arthritis, and enteropathic arthritis. Each has a variable propensity for oligoarthritis, uveitis, dactylitis, and skin involvement.

*One potential error with a spondyloarthropathy is not identifying an acutely inflamed eye as anterior uveitis or iridocyclitis.* Anterior uveitis has a prevalence of 25% with ankylosing spondylitis. There is no correlation between eye involvement and intensity of articular inflammation. Uveitis is usually acute and unilateral but may be recurrent with alternating eye involvement. Consensual photophobia (pain in the affected eye by illuminating the opposite eye), blurred vision, and lacrimation are typical.

Prompt treatment helps prevent complications (posterior synechiae, glaucoma).

## KEY POINTS

- Perform arthrocentesis in a suspected rheumatoid flare when one joint is involved out of proportion to the others or when primarily larger joints are involved.
- Consider cervical spine involvement when RA patients present with atraumatic neck pain.
- Obtain an MRI when neck pain in the RA patient is associated with myelopathic features, occipital headache, or with long-standing disease producing severe peripheral joint damage.
- Carefully evaluate an acutely painful red eye for anterior uveitis in patients with spondyloarthropathies.

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# SECTION XIII

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## NEURO

## AN UPDATE ON IDIOPATHIC INTRACRANIAL HYPERTENSION

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**EVELYN LEE, MD AND RAMIN TABATABAI, MD**

Idiopathic intracranial hypertension (IIH), previously known as benign intracranial hypertension, pseudotumor cerebri, and meningitis serosa, is typically a disease of overweight females who present with unrelenting headaches due to increased intracranial pressure (ICP). There is a strong association with oral contraceptive pills.

One relatively new pitfall in the diagnosis of IIH lies with our evolving understanding of its pathophysiology. IIH appears to exist on a spectrum of disease together with cerebral venous thrombosis (CVT). Emergency physicians are now caught in the diagnostic loop of patients with persistent headache, papilledema, raised ICP, and normal cerebrospinal fluid (CSF) who require a venogram by either magnetic resonance (MR) or computed tomography (CT) to exclude CVT as a diagnostic possibility.

IIH is defined as an elevation of ICP ( $>20$  cm H<sub>2</sub>O), with a normal CSF profile, and in the absence of space-occupying lesions (the modified Dandy diagnostic criteria). The typical patient is an obese woman aged 18 to 45 years old who presents with headaches, transient visual obscurations, pulsatile tinnitus, visual loss, and/or diplopia. There is also an association with rapid weight gain in the nonobese. Some additional established associations include prothrombotic disorders, polycystic ovarian syndrome, and, interestingly, both anemia and polycythemia. Medications linked to IIH include vitamin A, tetracycline, lithium, synthetic growth hormones, and oral contraceptives. The specific mechanism of increased ICP is not completely understood but may include overproduction or decreased absorption of CSF, venous stenosis, and thrombosis. Many patients with IIH are subsequently diagnosed with hypercoagulable states.

A detailed neurologic examination is indicated. The hallmark physical finding is papilledema or optic disc swelling on fundoscopic exam, a result of prolonged elevated ICP. One important note: elevated ICP can cause cranial nerve palsies; in particular the sixth cranial nerve (abducens) is commonly affected. This may result in diplopia with lateral gaze, a false localizing sign.

A CT head is done prior to lumbar puncture to identify any space-occupying lesions, hemorrhage, or hydrocephalus that could be causing the headaches or visual symptoms. Diagnosis of IIH is then made after a lumbar puncture shows normal CSF and an opening pressure of  $>20$  cm H<sub>2</sub>O. MR venography is the preferred study of choice to further evaluate for possible CVT or stenosis, most commonly found in the transverse sinuses.

Treatment for IIH focuses primarily on preserving or reversing vision loss through weight loss and medical management. Even a 5% to 10% weight loss can improve both headaches and visual disturbances. Medical management utilizes oral acetazolamide, a carbonic anhydrase inhibitor that theoretically reduces the secretion of CSF from the carbonic anhydrase-rich choroid plexus. The benefit of acetazolamide in high doses to assist with treating vision loss was demonstrated recently in a large, randomized, double-blind clinical trial performed by the Neuro-Ophthalmology Research Disease Investigator Consortium (NORDIC).

Surgical options such as optic nerve sheath fenestration (ONSF), CSF shunting, and venous sinus stent placement are options reserved for those who have failed medical management or suffer worsening vision loss secondary to papilledema.

## KEY POINTS

- Consider IIH in overweight females with headaches and visual disturbances.
- Although CT imaging is important initially to rule out life-threatening lesions prior to lumbar puncture, most patients will require an MR Venogram for further evaluation, notably to rule out CVT.
- The diagnosis of IIH is confirmed by a lumbar puncture demonstrating normal CSF and an opening pressure of  $>20$  cm H<sub>2</sub>O.
- The treatment of IIH is focused on weight loss and medical management with high-dose acetazolamide to prevent or reverse vision loss.

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# **NORMAL DIAGNOSTIC STUDIES DO NOT RULE OUT SHUNT MALFUNCTION**

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**AMY BUCKOWSKI, MD**

Placement of cerebrospinal fluid (CSF) shunts is the most common neurosurgical procedure performed on children in the United States. CSF shunts are designed to drain CSF from the central nervous system (CNS) into other parts of the body (peritoneal space, pleural space, right atrium, gallbladder, or ureter) to maintain a normal intracranial pressure. Unfortunately, up to 50% of CSF shunts will require revision within the first year of placement. Shunts may malfunction via infection, obstruction, migration, fracture/disconnection, or overdrainage. More than half of shunt infections occur within the first month of placement.

Shunt malfunction may present with symptoms of increased intracranial pressure such as headache, nausea, vomiting, lethargy, and altered mental status. Due to the distal drainage sites of CSF shunts, patients may also present with abdominal pain, shortness of breath, and symptoms specific to infection or perforation of the distal site. Fever may accompany CSF shunt infections, but the typical pathogens have low virulence and often cause indolent infections without fever or neurologic changes. Initial symptoms may be vague, and it is important to detect problems with CSF shunts before intracranial pressure elevation or shunt infection progresses.

Diagnosis of CSF shunt malfunction and infection requires a high degree of clinical suspicion because imaging and laboratory data may be falsely negative or equivocal. Early infection with skin flora is the most common cause for CNS shunt infection. To evaluate for shunt infection, ventricular



fluid should be sampled directly from the shunt, as lumbar puncture is not sensitive for detecting shunt infection. Although leukocytosis ( $\text{WBC} > 100/\text{mm}^3$ ) and elevated neutrophils (neutrophils  $> 10\%$ ) in the ventricular fluid are specific for shunt infection, these findings may be absent in up to 20% of shunt infections that grow positive cultures. Likewise, a negative ventricular fluid Gram stain does not rule out infection. It is important to understand the limitations of shunt fluid analysis because, if unrecognized, shunt infection can progress to cause shunt failure, ventriculitis, meningitis, or encephalitis, all of which convey high morbidity and mortality.

Radiographic imaging for detection of shunt malfunction typically includes a plain film shunt series and a head computed tomography (CT) or magnetic resonance (MR) study. The utility of the shunt series in the emergency department (ED) is questionable, with a sensitivity of only 11% to 30% for detecting shunt failures requiring surgical revision. Several studies have also observed that in the presence of a normal head CT, a shunt series changed the decision for surgical intervention in only 1% of cases. Nonetheless, a shunt series can be valuable in the early evaluation of shunt disconnection, kinking, or migration. Head CT or MR is used in combination with the shunt series to observe the appearance of the ventricles. Children with CSF shunts often have an abnormal-appearing CT or MRI at baseline, so studies should be compared to prior imaging. Ventricle size tends to decrease after CSF shunt placement and stabilizes by 12 months, so any ventricular enlargement compared to a prior study is likely abnormal.

Brain MR is an acceptable alternative to CT and should be used when available to reduce exposure to ionizing radiation. However, some CSF shunts use a magnetic mechanism to control CSF drainage, and MR may alter the shunt valve pressure setting. It is important to know the patient's shunt type or have a neurosurgeon involved prior to performing the MR.

As with ventricular fluid analysis for detecting shunt infection, imaging for detecting shunt malfunction is not sensitive enough to rule out shunt pathology. Even when performed together, the shunt series and head CT can miss a shunt obstruction in one in every eight children. Radiographic studies are even less sensitive for shunt infection. There is a significant risk of missing CSF shunt malfunction and infection when basing the diagnosis on studies performed in the ED. Diagnostic tests may increase the suspicion for shunt pathology, but do not rule it out. If shunt pathology is suspected but initial diagnostic studies are negative, a neurosurgeon should be involved in determining the need for further testing, treatment, and disposition.

## KEY POINTS

- Early symptoms of CSF shunt malfunction and infection may be vague, and it is important to make the diagnosis before progression of intracranial pressure elevation or shunt infection.
- Shunt fluid analysis may miss up to 20% of culture-positive shunt infections.
- Combined imaging (CT head and shunt series) may miss up to 12% of shunt malfunctions and is even less sensitive for detecting shunt infection.
- Neurosurgical consultation is appropriate if shunt malfunction is suspected even if the initial studies are negative.

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# DON'T BE FOOLED INTO ERRONEOUSLY DIAGNOSING PERIPHERAL VERTIGO

DANIEL MINDLIN, MD

Vertigo, the illusory sensation of physical movement, is a difficult presenting complaint for many emergency physicians, and rightly so. Not only can acute vestibular syndrome (AVS) be challenging to treat, it can be difficult to distinguish emergent and life-threatening causes from more benign ones.

The traditional and generally accepted approach to AVS is to divide its causes into central and peripheral etiologies (see [Table 185.1](#)), though causes less easily classified under this system do exist (e.g., medication effects, polypharmacy, and overdose). Of note, this approach begs the question of whether a given patient is in fact experiencing vertigo, rather than another form of “dizziness” such as presyncope or disequilibrium. Once it is established that vertigo is in fact the actual symptom, focus turns to ruling out vascular and other central causes of vertigo—specifically, vertebrobasilar insufficiency and stroke.

**TABLE 185.1 CAUSES OF ACUTE VESTIBULAR SYNDROME**

| CENTRAL                                | PERIPHERAL                                  |
|--|---|
| Vertebrobasilar ischemia or infarction | Benign paroxysmal positional vertigo (BPPV) |
| Cerebellar hemorrhage                  | Ménière disease                             |
| CNS tumor                              | Labyrinthitis                               |
| Increased intracranial pressure        | Vestibular neuritis                         |
| Migraine                               | Vestibular neuroma (Schwannoma)             |
|  | Perilymphatic fistula                       |

Unfortunately, computed tomography (CT) is insufficiently sensitive to reliably rule out most potentially catastrophic central diagnoses. Even magnetic resonance (MR) imaging can be negative early in the course of posterior circulation strokes; thus, it is essential that the clinician be comfortable with the clinical evaluation. [Table 185.2](#) summarizes the traditional distinction drawn between the clinical features of central and peripheral causes of vertigo.

| TABLE 185.2 CENTRAL VERSUS PERIPHERAL VERTIGO                                     |                     |                      |
|---|---------------------|----------------------|
|   | CENTRAL             | PERIPHERAL           |
| Symptom onset   | Gradual, continuous | Sudden, intermittent |
| Nystagmus direction   | Vertical            | Horizontal           |
| Nystagmus latency   | Short               | Long                 |
| Nystagmus fatigability  | No                  | Yes                  |
| Cerebellar symptoms<br>(diplopia, dysphagia,<br>disequilibrium)                   | Present             | Absent               |
| Cerebellar signs (dysmetria,<br>dysarthria, dysdiadochokinesia,<br>unsteady gait) | Present             | Absent               |

A new tool, the “HINTS” examination, has gained popularity in the quest to improve diagnostic accuracy in patients with vertigo. It aims to detect cases of stroke and vascular insufficiency that would otherwise be missed in patients presenting with isolated vertigo. “HINTS” is a clinically practical mnemonic for *head impulse* testing (“*HI*”), assessment for central (vertical or bidirectional) *nystagmus* (“*N*”), and *test of skew* (“*TS*”), by “cover-uncover” testing. The HINTS test, when performed by an experienced and knowledgeable provider, appears to outperform MR.

It is crucial to be aware of one potential stumbling block in the HINTS test: while absent central nystagmus and absent skew (i.e., negative findings)

are reassuring, it is a *normal* head impulse test in the setting of AVS that is highly concerning for a central etiology. This initially sounds counterintuitive, but because the vestibulo-ocular reflex is peripherally mediated, abnormalities in head impulse testing suggest a *peripheral* lesion, and thus, a *positive* result is actually favorable. When used appropriately, the HINTS exam can be a powerful tool to supplement routine bedside clinical evaluation in the patient with vertigo.

The Dix-Hallpike maneuver is a widely used test in the clinical examination of the patient with vertigo. Generally speaking, a positive Dix-Hallpike, when performed and interpreted correctly, is suggestive of benign paroxysmal positional vertigo (BPPV). However, this too can be falsely reassuring, as the positional changes of the Dix-Hallpike can provoke worsening vertigo and nystagmus even in central AVS, which can also lateralize to some extent. When performed in poorly selected patients—that is, frail elderly patients or those with presentations inherently suggestive of a central etiology, such as truncal ataxia or, notably, continuous rather than intermittent vertigo—the Dix-Hallpike maneuver can thus lead to an erroneous diagnosis of BPPV.

In general, the presence of auditory abnormalities—classically tinnitus or difficulty hearing—is typical of peripheral lesions. However, it is important not to exclude the presence of stroke solely on this basis. Medullary infarcts classically produce a “roaring” sound in the ipsilateral ear. Lateral pontine strokes, most often due to anterior inferior cerebellar artery (AICA) occlusion, frequently affect the vestibulocochlear nuclei, so the presence of disordered hearing alone cannot reliably exclude stroke. Of particular concern, blood flow to the inner ear is provided by the internal auditory artery, a branch of the AICA, and reduced perfusion quickly produces marked auditory dysfunction and AVS; thus, inner ear dysfunction due to labyrinthine infarction can be a sensitive early sign of impending AICA territory infarction. Labyrinthine infarction bears a disconcerting similarity to the typical presentation of Ménière disease, that is, intermittent vertigo associated with tinnitus or hearing loss. This underscores the importance of considering age, comorbidities, and other stroke risk factors in the complete evaluation of the patient with AVS. A low threshold for CT or MR angiography of the brain and neck is reasonable in appropriately selected patients, as MR (without angiography) of the brain can be normal early in the course of stroke or with isolated labyrinthine infarction.

Adherence to the basic tenets of AVS evaluation can prevent misdiagnosis of patients with vertigo. By remembering these points—that a *normal* head impulse test can suggest *central* vertigo, that the Dix-Hallpike maneuver can be abnormal in central vertigo, and that pontine stroke or

labyrinthine infarction can produce hearing abnormalities that may mimic Ménière disease—one can hope to avoid missing devastating central vascular events when they present to the ED.

## KEY POINTS

- A “normal” head impulse test in the context of AVS is worrisome—it suggests a central cause (e.g., stroke).
- In patients at high risk for stroke, do not be falsely reassured by a seemingly positive Dix-Hallpike maneuver.
- Auditory abnormalities do not reliably indicate a peripheral cause of vertigo.

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# **DIAGNOSING CERVICAL ARTERY DISSECTION IN THE ED: A REAL PAIN IN THE NECK!**

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**ERIK R. HOFMANN, MD, MS AND RAMIN TABATABAI,  
MD**

While the incidence of cervical artery dissection (CAD) is low, missing the diagnosis in the emergency department (ED) can be devastating. Two subtypes of CAD include carotid artery dissection and vertebral artery dissection, both of which are caused by damage to the intimal arterial walls. Carotid artery dissection is the underlying cause of 2.5% of all strokes and up to 20% of strokes in children and young adults. The annual incidence of vertebral artery dissection is far less common than carotid artery dissection, occurring in as few as 1 in 100,000 patients.

CAD is often classified by its mechanism, either traumatic or spontaneous in origin. The term spontaneous CAD is somewhat of a misnomer because most instances of “spontaneous” CAD are also believed to be associated with minor trauma in patients with an underlying predisposition. Therefore, a more appropriate term of “provoked spontaneous” CAD has been suggested, with the differences between these categories being somewhat artificial.

Mechanical stressors that have been implicated in CAD include blunt neck trauma or any minor manipulation of the neck including vomiting, painting a ceiling, practicing yoga, chiropractic neck manipulations, or even sneezing. Approximately 15% to 20% of patients will also have an underlying arterial abnormality such as fibromuscular dysplasia, Marfan syndrome, autosomal polycystic kidney disease, or osteogenesis imperfecta.

Regardless of mechanism, CAD occurs as a result of damage to the arterial wall and can lead to thrombus formation, causing the potential for thromboembolic stroke. In the case of carotid artery dissection, patients can present with typical anterior circulation stroke symptoms (e.g., hemiparesis, dysarthria) or other symptoms such as Horner syndrome and amaurosis fugax. In contrast, patients with vertebral artery dissection can present with a posterior circulation stroke syndrome (e.g., cranial nerve, cerebellar and long tract findings).

CAD is considered one of the great mimics in the ED because of its variable presentation. Because most patients with CAD do not present with dramatic neurologic findings, significant delays to diagnosis are common. Nonspecific symptoms in patients with carotid artery dissection include anterolateral neck pain, facial pain, and headache. In vertebral artery dissection, patients often present with posterior neck pain or occipital headache. In the absence of associated stroke, one could easily overlook a patient with isolated headache, facial pain, or neck pain.

## **CAROTID ARTERY DISSECTION**

Let's first discuss the more easily discernible presentations of CAD, those that present with findings of both pain and neurologic symptoms. As mentioned previously, the combination of carotid artery-specific focal neurologic deficits along with neck pain (in 25% to 50% of patients), facial pain (50%), or headache (44% to 69%) should alert the clinician toward the diagnosis of carotid artery dissection. The neck pain in carotid artery dissection tends to be ipsilateral and is located in the upper anterolateral cervical region, while the facial pain is most typically in the orbital region. The headache can be frontal, frontal-temporal, frontal-parietal, or rarely present as posterior cephalgia. The headache is most often gradual and throbbing but can also present as sudden and severe in onset. The following combination of pain and neurologic symptoms should immediately raise suspicion for carotid dissection:

- Pain + anterior cerebral ischemia
- Pain + complete or partial Horner syndrome
- Pain + cranial nerve palsies
- Pain + pulsatile tinnitus

The classic triad described for carotid artery dissection of headache, anterior cerebral stroke, and Horner syndrome is present in only 8% of patients. The most common neurologic deficits in these patients are either



anterior cerebral or retinal ischemic defects reported in 50% to 95% of patients. Amaurosis fugax, or transient monocular vision loss, occurs in ~3% of patients with carotid artery dissection and generally precedes the cerebral infarct. Dissection of the arterial wall can also lead to compromise of ascending sympathetic fibers causing a complete or incomplete Horner syndrome and can be identified in up to half of patients. Cranial nerve palsies occur in about 12% of patients, with the hypoglossal (XII) nerve most frequently affected. Isolated dysgeusia, although rare, can be highly suggestive of carotid artery dissection. Multiple cranial nerve findings can also occur simultaneously. Finally, pulsatile tinnitus has been reported, and a cervical bruit can sometimes be heard on exam.

## VERTEBRAL ARTERY DISSECTION

When evaluating for potential vertebral artery dissection, the clinician must consider an altogether different constellation of pain and neurologic symptoms than for that described in the carotid arteries. The head and neck pain is posterior as are the accompanying neurologic signs. Neurologic deficits by compressive aneurysmal dilation can present as unilateral radicular weakness in the C5–C6 distribution. Posterior circulation deficits can include ataxia, diplopia, dysarthria, locked-in syndrome, and lateral medullary syndrome.

## PATIENTS WITH NO NEUROLOGIC FINDINGS

In the most challenging of clinical circumstances, the patient may present with only headache, facial pain, or neck pain without neurologic findings. In this circumstance, the clinician has the arduous task of deciding when to obtain advanced imaging with angiography. Because current therapy for CAD involves anticoagulation to prevent thromboembolism, making the diagnosis at this early stage represents an opportunity to forestall a bad outcome. In these cases, the only clue of CAD may be the new onset of headache in a typical distribution, a concerning mechanism (often minor!) preceding the symptoms or the suggestion of a connective tissue disease. [Table 186.1](#) summarizes the clinical features of both types of CAD.

|  |
|--|
| <b>TABLE 186.1 CLINICAL FEATURES OF CERVICAL ARTERY DISSECTION</b> |
|--|

|                                | <b>CAROTID ARTERY<br/>DISSECTION</b>  | <b>VERTEBRAL ARTERY<br/>DISSECTION</b>   |
|--------------------------------|---|--|
| <b>Pain symptoms</b>           | Frontal headache<br>Facial or orbital pain<br>Anterior or lateral neck pain   | Occipital headache<br>Posterior neck pain  |
| <b>Neurologic<br/>symptoms</b> | Anterior circulation stroke<br>Partial or complete Horner<br>syndrome<br>Amaurosis fugax<br>Cranial nerve palsies<br>(including tongue deviation<br>and isolated dysgeusia)<br>Pulsatile tinnitus | Posterior circulation stroke<br>■ Hemianopsia<br>■ Ataxia<br>■ Diplopia<br>■ Dysarthria<br>■ Locked-in syndrome<br>■ Lateral medullary syndrome<br>Upper extremity radiculopathy |

## KEY POINTS

- Consider CAD in patients with a new onset of headache after blunt neck trauma or any activity that causes neck manipulation.
- CAD can present with or without neurologic deficits.
- Carotid artery dissection can present with any combination of head/facial/neck pain +/- anterior circulation stroke symptoms, Horner syndrome, amaurosis fugax, cranial nerve palsies, or pulsatile tinnitus.
- Vertebral artery dissection can present with any combination of head/neck pain + posterior circulation stroke symptoms.

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# **POSTERIOR CIRCULATION ISCHEMIC STROKE: IF YOU DON'T THINK ABOUT IT, YOU'LL MISS IT**

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**CRAIG TORRES-NESS, MD, MPH**

You walk into the room with a patient with multiple chief complaints that include nausea, vomiting, and occasional dizziness when she walks. The latter symptom of dizziness is in the nursing notes but doesn't come up in your interview. You thoroughly assess her, employing a broad nausea and vomiting differential, and determine that she has a benign abdominal exam, has a brown stool in her vault, and is tolerating some water and crackers. You rule out any surgical abdominal causes of her presentation, call it viral gastritis, and quickly provide the patient's disposition. Then, as you watch the patient leaving the emergency department (ED), you notice she is having difficulty ambulating and has a slightly widened gait. Was your diagnosis correct? Did you rule out all emergent causes of this presentation? If you did not think about or entertain a posterior circulation stroke, you may have just missed it. As the old adage states, you cannot diagnose what is not on your differential.

Ischemic stroke is defined as an acute occlusion of an intracranial vessel, resulting in reduced blood flow to the area supplied by that vessel. When cerebral blood flow falls to zero, infarction of brain parenchyma occurs within 4 to 10 minutes. A majority of these strokes involve the internal carotid arteries and their branches. Depending on the size of the infarct and duration of the occlusion, these patients present in a typical manner: unilateral weakness, facial droop, and aphasia. Helpful tools, such as the NIH Stroke Scale, have been developed to help identify such cerebrovascular events. Although most ischemic strokes occur in the anterior circulation,

~10% to 20% occur posteriorly, requiring us to be familiar with posterior circulation stroke symptoms. Acute occlusion of the posterior circulation, which arises from the vertebrobasilar system off of the subclavian artery, presents in a much less typical way and rarely presents with a single symptom. As a result, strokes involving the posterior circulation can be easily missed when they are not on the clinician's differential. The cost of missing such a stroke can be catastrophic for the patient. Cerebral edema from stroke in the posterior fossa can quickly lead to obstructive hydrocephalus, herniation, and death.

Strokes increase with age, male gender, atherosclerotic disease, atrial fibrillation, cigarette smoking, and alcohol abuse. Additionally, due to an aging population within the United States, the incidence of acute ischemic stroke is expected to increase drastically over the next 15 years. As a result, it is important that we remain vigilant when working up patients with those risk factors and to recognize symptoms that may not fit a classic stroke picture. Symptoms of a posterior circulation stroke include vertigo, imbalance, unilateral sensory or motor deficit, double vision, nausea, vomiting, and headache. Bulbar symptoms such as dysphagia, dysphonia, and dysarthria can also occur with both vertebral and basilar artery occlusions. One study found that the most common presentation of posterior stroke was nausea, vomiting, and truncal ataxia. Keep in mind that none of those three findings register on the NIH Stroke Scale!

A patient with risk factors and the aforementioned symptoms warrants a thorough neurologic evaluation including a complete examination of the cranial nerves. In addition to the routine assessment for motor and sensory weakness, all patients should also be tested specifically for signs of limb ataxia and cerebellar dysfunction. Testing for limb ataxia involves checking for dysmetria via a finger-to-nose test or a heel-to-shin test as well as evaluation for dysdiadochokinesia via rapid alternating hand movements. An important, and often neglected, component of the neurologic exam is ambulation to evaluate for truncal ataxia. Evidence of a wide-based or unsteady gait may be the patient's only abnormal neurologic finding. Ocular testing should include a check for nystagmus, gaze evaluation, and a confrontational field examination to look for signs of homonymous hemianopsia. In patients with isolated vertigo, by including head impulse testing, evaluating for bidirectional or vertical (e.g., central) nystagmus and ocular test of skew, a skilled clinician can achieve a sensitivity and specificity of >95% in identifying posterior circulation stroke. This combination of maneuvers is known by the mnemonic HINTS (see also [Chapter 185](#)).

Patients with posterior circulation strokes routinely experience delays in

care when compared to patients with anterior circulation strokes. This applies both to their evaluation by a neurologist and, when appropriate, the administration of fibrinolytic therapy. As a result, it is important that patients presenting with vague symptoms but with significant risk factors be evaluated thoroughly. A thorough neurologic exam, with special attention to cranial nerves, limb ataxia testing, gait evaluation, and the HINTS exam can all help reduce delays in diagnosis. Regarding our case presentation above, the provider would have done well to ambulate the patient prior to providing her discharge paperwork. Oh, and please don't forget to read the nursing notes!

## KEY POINTS

- Be aware of stroke risk factors and consider an extended neurologic exam in such patients, keeping in mind that nearly 20% of all ischemic strokes are posterior.
- Nausea, vomiting, and subtle neurologic deficits are concerning for posterior circulation stroke.
- Physical examination should include a complete neurologic exam including testing for dysmetria, dysdiadochokinesia, and gait.

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# **UNDERSTAND THE UTILITY AND LIMITATIONS OF DIAGNOSTIC IMAGING IN NONTRAUMATIC SUBARACHNOID HEMORRHAGE**

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Headache is the chief complaint for 3% of emergency department (ED) patients each year. Only 1% of those will ultimately be diagnosed with a nontraumatic subarachnoid hemorrhage (SAH). The diagnosis of SAH is challenging but essential in the ED as the disease is potentially catastrophic if not accurately identified. On the first day of illness, patients with nontraumatic SAH have a 12% mortality. This increases to a whopping mortality of 40% by 1 month after the event. To ensure appropriate diagnosis of this life-threatening disease, we should understand the utility and limitations of the various diagnostic imaging modalities currently employed in the patient with suspected SAH.

Fortunately, the combination of negative noncontrast head CT followed by negative lumbar puncture (LP) adequately rules out SAH. Subarachnoid blood seen on CT or a cerebrospinal fluid (CSF) specimen sample with either nonclearing red blood cells or xanthochromia is highly suggestive of SAH.

Noncontrast CT has high sensitivity for ruling out SAH when obtained very early after the event but drops rapidly with time. A pair of recent randomized controlled trials found a nearly 100% sensitivity in ruling out SAH with only a negative noncontrast CT head; however, it was important that the following criteria were met: (1) the headache must have clearly started <6 hours ago, (2) the CT used was at least a 3rd-generation

multidetector scanner, and (3) the read was finalized by an attending radiologist (either a neuroradiologist or a general radiologist with routine exposure to CT head). It should also be appreciated that these data only applied to patients with the chief complaint of headache. The authors of these studies suggest that a noncontrast CT head may thus be sufficient to rule out SAH when obtained within 6 hours of headache onset. The use of noncontrast CT head after 6 hours, however, will continue to necessitate an LP.

In recent years, some physicians have advocated for the use of CT angiography (CTA) in lieu of the traditional noncontrast CT/LP approach. CTA clearly has some desirable characteristics to support its use: it is fast, easy to obtain, and minimally invasive. Moreover, CTA can be quite sensitive in ruling out aneurysms >3 mm. However, the strategy of utilizing CTA in place of CT/LP does pose some fundamental pitfalls that we should recognize. These include an increased exposure to ionizing radiation, contrast exposure with possible reactions, and, perhaps most importantly, the diagnosis of incidental (asymptomatic) aneurysms. The prevalence of aneurysms in the general population is quite high (~3%), while the incidence of SAH is exceedingly low (0.01%). Therefore, a majority of aneurysms detected on CTA are *incidental*—they pose little or no risk of rupture. Simply identifying these “nonculprit” aneurysms can pose potential downstream harm to the patient in the form of unnecessary neurosurgical consultation, further imaging, and the potential for unnecessary surgical or invasive repair. Although obtaining a CTA may ultimately be less labor intensive in the ED and tempting to obviate the need for LP, the potential harm with this approach must be appreciated.

Magnetic resonance angiography (MRA) is another diagnostic modality becoming increasingly more available in the ED. Although the image quality may provide a greater ability to evaluate other potential causes for headache, the same inherent risks and limitations exist as discussed above for CTA, namely, the identification of incidental aneurysms. Both CTA and MRA can be valuable diagnostic tools for the neurosurgeon once SAH has been diagnosed via noncontrast CT and/or LP; neither, however, are recommended routinely for the initial evaluation of headache.

Currently, both the American Heart Association (AHA) guidelines on management of SAH and the American College of Emergency Physicians (ACEP) Clinical Policy on the evaluation and management of acute headache advocate the use of noncontrast head CT followed by LP for the initial evaluation of suspected SAH. Though newer diagnostic modalities are now more readily available, awareness of the pitfalls discussed here is warranted when departing from published guidelines.

## KEY POINTS

- Guidelines continue to recommend the combination of noncontrast CT head followed by LP to adequately rule out SAH.
- A noncontrast head CT may rule out SAH when obtained within 6 hours of headache onset.
- CTA and MRA are not recommended as part of the routine initial workup as they may expose the patient to unnecessary risks.

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## DON'T FORGET ATYPICAL CAUSES OF STATUS EPILEPTICUS!

R. JAMES SALWAY, MD

The definition of status epilepticus has evolved as understanding of its potential sequelae has increased. Before examining the different causes of generalized convulsive status epilepticus (GCSE), it is important to first discuss this changing definition. Previously defined as any seizure activity lasting for more than 30 minutes, most neurologic organizations and trials are now defining GCSE as two seizures or more without return to neurologic baseline OR any seizure lasting more than 5 minutes. This updated definition reflects a new recognition of the increased morbidity and mortality associated with prolonged seizure activity. Some studies have shown a 30-day mortality in adults with GCSE of 19% to 27%. Importantly, mortality has been shown to correlate directly with the duration of the seizure. These facts underscore the importance of rapidly identifying the underlying etiology of GCSE.

While nonadherence with medications in patients with known seizures remains the most likely underlying cause of GCSE, up to *50% of GCSE patients have no prior seizure history*. This means that other causes of GCSE must be considered up front when we are dealing with seizures that are refractory to standard treatment. The easiest way to approach these atypical causes of GCSE is to break them down into broad categories: infectious, metabolic, drug related, and other. Treatment and diagnostic considerations for each of these categories are outlined in [Table 189.1](#).

**TABLE 189.1 CAUSES, DIAGNOSIS, AND TREATMENT OF STATUS EPILEPTICUS**

| ETIOLOGY                | DIAGNOSIS                       | TREATMENT  |
|-------------------------|---------------------------------|--|
| <b>Infectious</b>       |                                 |  |
| Meningitis/encephalitis | CT/LP                           | IV antibiotics, antivirals, antifungals  |
| <b>Metabolic</b>        |                                 |  |
| Hypoglycemia            | Rapid glucose test<br><80 mg/dL | 50 mL of D50 in adults<br>Consider 100 mg of thiamine<br>(in alcoholics, malnourished)<br>Lower concentrations in children and infants<br>(2 mL/kg of D25 in children, 5 mL/kg of<br>D10 in infants) |
| Hyponatremia            | Chemistry                       | Hypertonic saline  |
| Hypernatremia           | Chemistry                       | Hypotonic solutions  |
| Hypocalcemia            | Chemistry                       | Appropriate electrolyte repletion  |
| Hypokalemia             | Chemistry                       | Appropriate electrolyte repletion  |
| Hypomagnesemia          | Chemistry                       | Appropriate electrolyte repletion  |
| <b>Drug Induced</b>     |                                 |  |
| Isoniazid               | History                         | Pyridoxine<br>(1 g per gram of INH or 5 g IV)  |
| Cyclic antidepressants  | History, EKG                    | Sodium bicarbonate   |
| Alcohol withdrawal      | History                         | Benzodiazepines  |
| <b>Other</b>            |                                 |  |
| Eclampsia               | Beta hCG, history               | Magnesium  |

Infectious etiologies such as meningitis and encephalitis can both cause GCSE. Fever and infection have been cited as the most common cause of seizures in children. Computed tomography (CT) of the head and lumbar puncture (once the patient has stopped seizing!) may be necessary to rule out CNS infection, especially in the setting of undifferentiated fever in an adult.

Metabolic disturbances are frequently responsible for GCSE. Hypoglycemia is one of the most common and readily diagnosed etiologies. Seizure activity can occur when blood glucose is below 45 mg/dL. A rapid bedside glucose test should be done as soon as possible. Both hyponatremia and hypernatremia are also common metabolic derangements that can lead to GCSE. A sodium level <120 mEq/L or >160 mEq/L should alert us to the diagnosis. Hypocalcemia, hypokalemia, and hypomagnesemia often occur concurrently and should be treated simultaneously.

A multitude of drugs can cause GCSE and the list is too extensive to be completely addressed here. Major culprits that should not be overlooked include isoniazid (INH), antidepressants (particularly tricyclic antidepressants, which are making a comeback), and lithium. Methamphetamine intoxication and alcohol withdrawal are also on the differential.

Lastly, there are many “other” causes of GCSE. In the young (possibly pregnant) woman with seizure, eclampsia should be considered, even in the postpartum period, as a significant number of cases occur in the weeks after delivery. In addition, trauma, stroke, neoplasm, and degenerative CNS diseases round out our list of “other” causes to consider.

When faced with GCSE, we need to think broadly and consider the wide range of possible etiologies beyond mere medication nonadherence. Many of these etiologies are rapidly reversible with treatments that can terminate further seizure activity and directly reduce morbidity and mortality.

## KEY POINTS

- GCSE is a serious disease process and should be terminated as soon as possible.
- Hypoglycemia is the most common metabolic cause of GCSE, followed by sodium imbalances.
- Don’t forget about eclampsia in the young seizing female patient, even in the postpartum period.
- Keep an open mind when presented with GCSE. Infectious, metabolic, drug-induced, and “other” atypical causes must be considered in the differential.

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# **LEAVE IT ALONE: BLOOD PRESSURE MEASUREMENT IN ISCHEMIC STROKE**

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**AMIR A. ROUHANI, MD**

Stroke is defined as any vascular injury that leads to a reduction of cerebral blood flow to an area of the brain resulting in neurologic impairment. Ischemic stroke accounts for 85% of strokes and can result from thrombosis, embolism, or systemic hypoperfusion. Hemorrhagic stroke, which includes intracerebral hemorrhage (ICH) and nontraumatic subarachnoid hemorrhage (SAH), accounts for the remaining 15%.

After an acute stroke, blood flow and therefore oxygen transport are reduced locally, resulting in hypoxia of the areas near the location of the original insult. Within the ischemic cerebrovascular bed, there are two major zones of injury: the core ischemic zone and the ischemic “penumbra.” The penumbra is the viable tissue immediately surrounding the irreversibly damaged ischemic core where distal branches become dilated and perfusion pressure is low. The goal of acute stroke management is to salvage this penumbral tissue and optimize resultant brain function.

A central concept in the medical management of all acute strokes has focused on the maintenance of cerebral perfusion pressure (CPP) in order to optimize perfusion to these zones of injury. Cerebral perfusion pressure (CPP) is derived by the following formula: CPP (cerebral perfusion pressure) = MAP (mean arterial pressure) – ICP (intracerebral pressure). Permissive hypertension involves the avoidance of aggressive lowering of blood pressure (BP) to maintain an elevated MAP and thus preserve CPP.

In contrast to hemorrhagic stroke, where there recently has been intense

focus on BP reduction in the emergency department (ED) phase of management (see [Chapter 193](#)), guidelines for ischemic stroke continue to emphasize “*permissive hypertension*”—the deliberate “hands-off” maintenance of BP in the ED.

Elevated arterial BP is a common occurrence at the time of presentation among patients with ischemic stroke, occurring in up to three-quarters of cases. This may be due to underlying chronic hypertension, an acute sympathetic response, or other stroke-related mechanisms. This initial hypertensive response is most pronounced immediately following the acute stroke. BP will generally begin to decrease spontaneously within 90 minutes and steadily decline over the first 24 hours.

Multiple studies have found a U-shaped relationship between the admission BP and poor clinical outcomes. Extreme arterial hypertension can be detrimental because it may lead to worsening cerebral edema and hemorrhagic transformation, encephalopathy, cardiac complications, and renal insufficiency. Moreover, extreme arterial hypotension can lead to decreased perfusion to multiple organs, especially the ischemic brain. Finally, there may be certain situations where concomitant medical conditions, such as myocardial ischemia, aortic dissection, and heart failure, may accompany acute ischemic stroke and may be exacerbated by arterial hypertension. Therefore, moderate arterial hypertension might represent the best opportunity to optimize CPP while avoiding the harm of extreme hypertension. As a result of this, current guidelines recommend that hypertension be “permitted” unless it is *extremely* high [systolic blood pressure (SBP) >220 mm Hg or diastolic blood pressure (DBP) >120 mm Hg].

The 2013 American Heart Association (AHA)/American Stroke Association (ASA) guidelines for the early management of patients with acute ischemic stroke call for permissive hypertension in patients who are not candidates for reperfusion therapy with fibrinolytic agents in order to maintain cerebral perfusion. Permissive hypertension involves no active attempts at lowering BP unless the patient has an SBP >220 mm Hg or DBP >120 mm Hg. BP control may also be initiated for patients with a concurrent medical condition in which a clinical benefit would be achieved with BP lowering. If BP lowering strategies are initiated, a suggested target is a 15% reduction in SBP for the first 24 hours. In patients who are eligible for treatment with intravenous fibrinolytic agents, BP should be carefully lowered so that SBP is <185 mm Hg and DBP <110 mm Hg. BP should be maintained below 180/105 mm Hg for at least 24 hours (see [Table 190.1](#)).

### **TABLE 190.1 GUIDELINES FOR THE EARLY MANAGEMENT OF PATIENTS WITH ACUTE ISCHEMIC STROKE: A GUIDELINE FOR HEALTH CARE PROFESSIONALS FROM THE AMERICAN HEART ASSOCIATION/AMERICAN STROKE ASSOCIATION (2013): CLASS I RECOMMENDATIONS<sup>A</sup>**

- In patients with markedly elevated blood pressure who do not receive fibrinolysis, a reasonable goal is to lower blood pressure by 15% during the first 24 h after onset of stroke. The level of blood pressure that would mandate such treatment is not known, but consensus exists that medications should be withheld unless the SBP is >220 mm Hg or the DBP is >120 mm Hg (Class I; Level of Evidence C). (Revised from the previous guideline)
- Patients who have elevated blood pressure and are otherwise eligible for treatment with intravenous rtPA should have their blood pressure carefully lowered so that their SBP is <185 mm Hg and their DBP is <110 mm Hg (Class I; Level of Evidence B) before fibrinolytic therapy is initiated. If medications are given to lower blood pressure, the clinician should be sure that the blood pressure is stabilized at the lower level before beginning treatment with intravenous rtPA and maintained at <180/105 mm Hg for at least the first 24 h after intravenous rtPA treatment. (Unchanged from the previous guideline)

<sup>a</sup> Class I (Strong): Conditions for which there is evidence, general agreement, or both that a given procedure or treatment is useful and effective.

*Source:* Jauch EC, Saver JL, Adams HP Jr, et al. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2013;44:870.

First-line agents for BP management in acute ischemic stroke include labetalol and nicardipine. Both allow rapid and safe titration to the goal BP. Labetalol may be started at 10 to 20 mg intravenously over 1 to 2 minutes and may be repeated once prior to initiating a continuous infusion of 2 to 8 mg/min. Nicardipine may be started intravenously at 5 mg/h, titrating up by 2.5 mg/h every 5 to 15 minutes (maximum 15 mg/h).

## **KEY POINTS**

- The management of elevated BP in acute ischemic stroke should follow the principle of permissive hypertension. For those who are not treated with thrombolytic therapy, antihypertensive therapy should only be considered if the hypertension is extreme (SBP > 220 mm Hg

or DBP > 120 mm Hg) or if the patient has another clear indication (active myocardial ischemia, congestive heart failure, or aortic dissection).

- When treatment is indicated, the goal should be to lower BP by ~15% during the first 24 hours after stroke onset.
- For patients with acute ischemic stroke who will receive thrombolytic therapy, antihypertensive treatment is recommended so that SBP is <185 mm Hg and DBP is <110 mm Hg. Labetalol and nicardipine are the recommended first-line agents.

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# CEREBRAL VENOUS SINUS THROMBOSIS: A RARE DIAGNOSIS WITH A COMMON CHIEF COMPLAINT

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Cerebral venous sinus thrombosis (CVST) is a serious yet uncommon cause of stroke, seizure, and headache that is often underrecognized and misunderstood. CVST accounts for ~0.5% to 1% of all strokes and results from thrombosis and obstruction of the cerebral venous dural sinuses. This disease appears to have a predilection for females of reproductive age and is often associated with risk factors such as oral contraception and pregnancy, though CVST can affect both men as well. Most patients are in the third or fourth decade of life, though 8% of patients diagnosed are over the age of 65. A thorough investigation for underlying prothrombotic pathology or family history of coagulopathy will reveal at least one risk factor in 85% of patients (see [Table 191.1](#)), but some patients, especially the elderly, may have no easily identifiable risks.

**TABLE 191.1 RISK FACTORS FOR CEREBRAL VENOUS SINUS THROMBOSIS**



*Transient risk factors*

Infection—CNS, ENT (mastoiditis, sinusitis), sepsis, TB

Pregnancy and puerperium

Head trauma

Lumbar puncture

Radical neck surgery

Neurosurgical procedures

Jugular and subclavian catheters

Drugs—OCPs, L-asparaginase, androgens, ecstasy, sildenafil

Diabetic ketoacidosis

*Permanent risk factors*

Genetic—protein C/S, antithrombin deficiencies, factor V Leiden

Acquired—antiphospholipid syndrome, nephrotic syndrome

Malignancy—meningioma, leukemia, lymphoma

Anemia—sickle cell, PNH, polycythemia, thrombocytopenia

Inflammatory disease—SLE, Sjögren, Wegener granulomatosis, inflammatory bowel disease, sarcoidosis

The mechanisms involved in CVST depend on the initial thrombotic event. An occlusive thrombus of the cerebral veins causes localized edema that is both cytotoxic from cell death and vasogenic from increased pressure in the venules. On the other hand, a thrombus in the sinuses causes a secondary increased venous pressure that will decrease CSF flow and lead to an increase in intracranial pressure (ICP). Ultimately, both forms of thrombus will lead to similar symptomatology by different pathways.

The single biggest challenge with CVST is its nonspecific early clinical course. On average, it takes 7 days after symptom onset to establish a definitive diagnosis. Headache is the most common symptom in these patients, occurring in 90% of patients. The headache may be the only symptom at first, preceding more specific or worrisome neurologic symptoms by several days. To further complicate matters, the headache presentation is variable, most commonly diffuse, and gradually worsening over weeks, but sometimes unilateral or sudden and severe in onset mimicking a subarachnoid hemorrhage. Fortunately, the majority of patients will present with papilledema.

A headache that is worsening when lying supine or worsening with Valsalva maneuvers should prompt further questioning into potential predisposing conditions as these can be signs of increased ICP. A seizure should prompt further evaluation as ~40% of patients with CVST will have either focal or generalized seizure activity—not to mention the need to consider other more common life-threatening diagnoses such as meningitis or a space-occupying lesion. Nevertheless, the diagnosis of CVST should

remain in the differential diagnosis during the workup, especially if the opening pressure on lumbar puncture exceeds 20 cm H<sub>2</sub>O.

A patient presenting with a combination of headache and visual symptoms such as horizontal diplopia and cranial nerve VI (abducens) palsy (secondary to increased ICP) should also prompt further questioning into potential risk factors for CVST. When papilledema exists, further imaging is also warranted to evaluate for possible CVST.

Severe cases of CVST can result from venous infarction with or without hemorrhage and cerebral edema. These patients may present with focal neurologic signs that do not follow a typical arterial distribution of stroke. The most common neurologic deficit is motor weakness, which can occur bilaterally or unilaterally. Other potential neurologic deficits include encephalopathy, cranial nerve palsies, and aphasia.

If suspicion is high enough, appropriate imaging can lead to definitive diagnosis and lifesaving treatment. Currently, the most sensitive technique for diagnosis is magnetic resonance (MR) imaging with venography (MRV). The combination of absence of blood flow with an abnormal signal in the sinus confirms the diagnosis of thrombosis. Several studies have evaluated the use of laboratory values, such as the D-dimer, to help with diagnosis of CVST, but, unfortunately, none has a low enough negative predictive value to rule out CVST.

Heparin therapy should be promptly started once thrombus is confirmed, even in the setting of venous hemorrhage, to decrease clot propagation and other complications. Heparin has been shown to be relatively safe even in patients with a hemorrhagic component in the setting of CVST. Patients will require admission, preferably to a neurosciences unit. Thrombectomy may be considered in aggressive deadly variants of CVST.

## KEY POINTS

- The diagnosis of CVST is frequently delayed, often with grave consequences.
- Risk factors include a history of prothrombotic disease, pregnancy, and the postpartum period.
- CVST may present as an isolated headache. Look for clinical features of increased ICP and papilledema to direct further investigation.
- MRV is the diagnostic imaging modality of choice.

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# **GREAT IMITATORS OF ACUTE STROKE**

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**AARTI JAIN, MD**

Stroke mimics, or nonvascular disorders that produce focal neurologic deficits, often confound the diagnosis of acute ischemic stroke. As many as 20% to 25% of suspected stroke presentations can be attributed to nonvascular disease processes. In an age of new interventional stroke therapies that include endovascular treatment and fibrinolysis, it is increasingly important to identify stroke mimics and avoid potential harm from secondary stroke prevention therapies. Common stroke mimics include, but are not limited to, Todd paralysis, hypoglycemia, cerebral neoplasm, hemiplegic migraine, and functional hemiparesis.

## **TODD PARALYSIS**

Todd paralysis is a transient neurologic condition that is most commonly characterized by focal paresis following a seizure. Reversible, nonstructural alterations in neuronal function that occur in the postictal period may cause focal neurologic deficits that simulate the presentation of acute stroke. While the duration of these deficits is generally transient, focal findings can last up to 1 to 2 days. Postictal weakness is primarily seen after partial motor seizures but can also follow generalized seizures. Often, this stroke mimic is only identified after witnessing further seizure activity or inquiring about a history of seizure disorder.

## **HYPOGLYCEMIA**

Hypoglycemia can be defined as a serum glucose level below normal and is

considered severe when levels fall below 45 mg/dL. It is the quintessential stroke mimic as it is the most rapidly identifiable and easily reversible nonvascular cause of neurologic deficits. Hemiplegia, cortical blindness, and aphasia have all been reported as common stroke-like presentations of hypoglycemia. While it is essential for physicians to obtain a rapid serum glucose level in any patient suspected of having an acute stroke, it must also be noted that neurologic deficits may occasionally persist for hours after IV glucose administration. Other metabolic imbalances such as hyperglycemia, hyponatremia, and hepatic encephalopathy can present similarly.

## **CEREBRAL NEOPLASM**

Though primarily indolent in presentation, intracerebral mass lesions can cause rapidly progressive visual changes, aphasia, and other focal neurologic deficits that mimic acute stroke. As many as 6% of patients with brain tumors present with symptoms that are <1 day in duration. This stroke-like clinical picture can be attributed to a variety of mechanisms including obstructive hydrocephalus, vascular compression, and tumor apoplexy.

## **HEMIPLEGIC MIGRAINE**

Primary headache disorders account for 9% of stroke mimics. One rare migraine variant, the familial hemiplegic migraine (FHM), is defined as migraine with aura consisting of fully reversible motor weakness. Autosomal dominant in inheritance, FHM most commonly affects young females and shows high familial penetrance. Though FHM is a diagnosis of exclusion, a careful family history or evidence of prior similar recurrent episodes may raise suspicion of this migraine subtype.

## **FUNCTIONAL HEMIPARESIS**

Conversion disorder, or functional neurologic symptom disorder, is characterized by neurologic symptoms that are inconsistent with a medical etiology but not intentionally produced by the patient. Weakness can be seen in up to 30% of these individuals, and though most commonly unilateral or hemiparetic, can also be bilateral or affect only a single limb. Key to identifying conversion disorder as a stroke mimic is an inconsistent exam, where the extent of the impairment is often found to be task dependent. On physical examination, Hoover sign, or weakness with hip extension in the affected leg that disappears during contralateral flexion of the unaffected leg

against resistance, can suggest a functional etiology of paresis.

Todd paralysis, hypoglycemia, cerebral neoplasm, hemiplegic migraine, and functional hemiparesis constitute just a few of the many disease processes that can present with a stroke-like clinical picture. Other stroke mimics include infection, syncope, peripheral vestibular disorders, dementia, and intoxication. Given the extensive list of stroke mimics as well as the diagnostic limitations in detecting ischemic strokes in the emergency department, it is important to keep a broad differential and perform a thorough history in any patient who at first glance appears to be suffering a stroke.

## KEY POINTS

- Twenty to twenty-five percent of suspected stroke presentations are attributable to nonvascular etiologies.
- In an era of invasive and potentially harmful secondary stroke prevention therapies, it is increasingly important to rapidly identify stroke mimics.
- Common stroke mimics include hypoglycemia, Todd paralysis, primary headache disorders, cerebral neoplasm, and functional hemiparesis.
- Obtain a rapid serum glucose level, and consider nonvascular disease processes in any patient with acute neurologic impairment.

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# BLOOD PRESSURE IN THE PATIENT WITH INTRACRANIAL HEMORRHAGE—BRING IT DOWN!

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**DANIEL R. RUTZ, MD AND EDWARD STETTNER, MD**

Spontaneous intracerebral hemorrhage (ICH) is the second most common type of stroke after ischemic stroke. Mortality is high, with up to half of all patients not surviving to 30 days. While to date no single intervention has been proven to reduce mortality in this devastating disease, early recognition and aggressive treatment in the emergency department (ED) is likely critical in the quest to reduce long-term sequelae. Initial resuscitation of ICH involves airway management, rapid neuroimaging, correction of coagulopathy, and early neurosurgical consultation. The role of blood pressure (BP) management remains controversial, but recent studies have led to new guidelines that reflect a change in our understanding of the underlying pathophysiology of ICH.

Patients with ICH often have coexisting high BP. Hypertension is both a cause of spontaneous ICH and a response to the physiologic insult. The latter is thought to be a result of increased sympathetic tone and increased intracranial pressure (ICP). Severe elevations in BP, which are typical, may worsen outcomes for patients through expansion of the hematoma via ongoing bleeding. Observational data have shown an association between patients with a systolic blood pressure (SBP) above 150 mm Hg in the first 12 hours after ICH and a twofold increase in the risk of death or dependency. Other studies have identified hematoma growth to be associated with substantially worsening outcomes. Given the substantial morbidity and mortality burden of ICH, there has been renewed focus on the efficacy of early BP control in the ED. [Table 193.1](#) summarizes the current BP

management guidelines (2015) of the American Heart Association/American Stroke Association for spontaneous ICH.

**TABLE 193.1 2015 GUIDELINES FROM THE AHA/ASA FOR BP MANAGEMENT IN ACUTE ICH**

1. For ICH patients presenting with SBP between 150 and 220 mm Hg and without contraindication to acute BP treatment, acute lowering of SBP to 140 mm Hg is safe (Class I; Level of Evidence A) and can be effective for improving functional outcome (Class IIa; Level of Evidence B). (Revised from the previous guideline.)
2. For ICH patients presenting with SBP > 220 mm Hg, it may be reasonable to consider aggressive reduction of BP with a continuous intravenous infusion and frequent BP monitoring (Class IIb; Level of Evidence C). (New recommendation) Recommendations are class C: SBP = systolic blood pressure; MAP, mean arterial pressure; CPP = MAP – ICP.

Many clinicians are concerned that lowering BP too rapidly in stroke can be dangerous. This is based on the concept that patients with chronic hypertension and cerebrovascular disease become dependent on higher than normal BP to provide adequate cerebral perfusion. This does indeed appear to be true in ischemic stroke, where a vulnerable area (ischemic penumbra) around the infarction is sensitive to a lowered BP. However, modern neuroimaging has demonstrated that ICH patients do not have an ischemic penumbra. It is this critical difference that seems to tip the balance toward a more aggressive approach to BP management in an attempt to prevent hematoma expansion and rebleeding.

The claim for the safety of aggressive BP reduction is supported by the Antihypertensive Treatment of Acute Cerebral Hemorrhage (ATACH) and Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage (INTERACT) clinical trials. The INTERACT study randomly assigned ICH patients to aggressive BP control (target SBP < 140 mm Hg in the first hour, maintained for 24 hours) compared to more lenient control (target SBP < 180 mm Hg in the first hour, maintained for 24 hours). This study demonstrated a trend toward lower relative and absolute growth of hemorrhage in the intensive treatment group compared with the control group. Importantly, there were comparable numbers of adverse events and similar patient functional outcomes.

The largest randomized trial to date involving early BP lowering in ICH, the INTERACT2 study, demonstrated improvement in functional outcomes in patients with aggressive BP control with no differences in mortality or major nonfatal adverse events. In INTERACT2, patients with ICH and SBP



between 150 and 220 mm Hg were randomized to intensive (target SBP goal <140 mm Hg within 1 hour of randomization, maintained for 7 days) versus lenient BP control (SBP goal < 180 mm Hg). Of note, this trial did not demonstrate significant reduction in the primary outcome of death or major disability (mRS score 3 to 5). However, an analysis of scores on the mRS for patients in the intensive group did show improved functional outcomes and patients in this group self-reported improved health-related quality of life metrics. Recent data from the ATACH-II clinical trial contradict the secondary benefits observed in INTERACT2. Patients in the ATACH-II were randomly assigned to BP reduction goals of 110 to 139 mm Hg (intensive BP lowering) or 140 to 179 mm Hg (standard care). The authors reported no significant difference in primary outcome, (mRS score 4 to 6 at 3 months), and, a higher incidence of serious adverse events within 3 months in the intervention group (25.6% in intervention, 20% in control;  $p = 0.05$ ). Data from these trials demonstrate the need for further research into the risks and benefits of aggressive blood pressure reduction below 140 mm Hg in patients with spontaneous ICH.

Medications used in the trials above included labetalol, nicardipine, hydralazine, nitroprusside, nitroglycerin, enalapril, and esmolol. The most commonly used were labetalol and nicardipine. Current guidelines do not support the use of one medication over another, but in general, intravenous (IV) titratable agents are required to provide the type of control necessary in ICH.

Be mindful that while mortality benefit has not been clearly delineated, sustained elevated BP is associated with worse functional outcomes for these patients. Further research is needed to examine the effect of BP lowering on hematoma growth and the effect of aggressive BP lowering below 140 mm Hg. Moreover, patients with the most severe BP elevations (SBP > 220 mm Hg) were excluded from the INTERACT2 trial—more data is needed to guide therapy in these patients.

## KEY POINTS

- ICH carries a significantly higher risk of mortality than does ischemic stroke.
- In contrast to ischemic stroke, initial BP reduction in ICH appears to be safe and may reduce hematoma expansion.
- In patients with ICH and elevated SBP between 150 and 220 mm Hg, reduction of BP in the ED with an IV titratable agent to a target of less

than 140 mm Hg is appropriate.

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## HOW TO DISPOSITION THE PATIENT WITH SUSPECTED TIA

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**ALLEN CHIOU, MD AND MINDI GUPTILL, MD, FACEP**

Transient ischemic attack (TIA) is a common life-threatening condition that occurs with an estimated annual incidence of 240,000 cases per year in the United States. It accounts for 0.3% of all emergency department (ED) visits. In the not so distant past, TIA was described as a sudden-onset, focal neurologic event of vascular origin lasting <24 hours. However, as imaging studies become more sensitive to detect tissue infarction, it has become apparent that many so-called TIAs are actually strokes. In fact, in some cases, infarction can be seen on diffusion-weighted MR (magnetic resonance) imaging after as few as 10 or 15 minutes of symptoms. The American Heart Association/American Stroke Association (AHA/ASA) currently defines TIA as a “*transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction.*” TIA is therefore an ischemic central nervous system (CNS) disease on the same spectrum as stroke, which is differentiated by the presence of tissue infarction.

Patients with a suspected TIA must have rapid and accurate ED evaluation. Although many patients may have a benign course of disease, there is a considerable short-term risk for stroke. Large cohort and population-based studies reported in the last several years have demonstrated a higher risk of early stroke after TIA than previously appreciated. Ten to fifteen percent of patients have a completed stroke within 3 months, with half occurring within 48 hours. Therefore, disposition of the patient with TIA requires careful risk stratification.

A widely used tool for stroke risk stratification after TIA is the ABCD<sup>2</sup> score, developed and validated in 2007 by Johnson and colleagues. The

ABCD<sup>2</sup> score is optimized for 2-day stroke risk prediction. ABCD<sup>2</sup> also predicts risk at 7 and 90 days after TIA. The scoring system assigns points based on age, blood pressure elevation, clinical features, duration of symptoms, and presence of diabetes (see [Table 194.1](#)). Patients in the initial study were categorized as high risk (score of 6 to 7), moderate risk (score of 4 to 5), or low risk (score of 0 to 3). The overall 2-day stroke risk for the high-, moderate-, and low-risk categories were 8.1%, 4.1% and 1.0%, respectively.

**TABLE 194.1 ABCD SCORING SYSTEM**

| <b>CRITERIA</b>             | <b>POINTS</b> |
|-----------------------------|---------------|
| Age > 60 y                  | 1             |
| Blood pressure              |               |
| Systolic >140 mm Hg or      | 1             |
| Diastolic > 90 mm Hg        |               |
| Clinical features           |               |
| Unilateral weakness         | 2             |
| Isolated speech disturbance | 1             |
| Other symptom               | 0             |
| Duration of symptoms:       |               |
| ≥60 min                     | 2             |
| 10–59 min                   | 1             |
| 0 min                       | 0             |
| Diabetes                    | 1             |

*Source:* Easton JD, Saver JL, Albers GW, et al. Definition and evaluation of transient ischemic attack. A scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. *Stroke*. 2009;40(6):2276–2293.

It is important to note that external validation of the ABCD<sup>2</sup> score has not been definitive. To date, the largest multicenter prospective validation study in 2011 by Perry et al., showed poor predictive value of the ABCD<sup>2</sup> score at 7 and 90-day intervals after TIA. A 2-day stroke risk assessment was not included in this study. Although the ABCD<sup>2</sup> score is used widely to risk stratify patients, it should not replace clinical judgment.

Currently the AHA/ASA recommends use of the ABCD<sup>2</sup> score and

states that hospital admission is reasonable if patients present within 72 hours of symptoms and any of the following criteria are present: ABCD<sup>2</sup> score of  $\geq 3$ , ABCD<sup>2</sup> score of 0 to 2 and uncertainty that diagnostic workup can be completed within 2 days as an outpatient, or ABCD<sup>2</sup> score of 0 to 2 and other evidence that indicates the patient's event was caused by focal ischemia.

The TIA workup as recommended by the AHA/ASA includes neuroimaging within 24 hours of symptom onset (preferably with diffusion-weighted brain MR), noninvasive imaging of the cervical and intracranial vessels (with Doppler ultrasound, computed tomography, or MR angiography), electrocardiogram, and routine blood tests. Continuing advances in neuroimaging will likely further refine the evaluation and management of TIA in the future.

The disposition and timing for the diagnostic workup of post-TIA ED patients should be based on clinical judgment and local resource availability. The treating clinician should take into account high-risk features such as the duration of symptoms, crescendo (or escalating) symptoms, and/or the presence of known pathology such as carotid artery stenosis, hypercoagulability, or a cardiac source of emboli. Additionally, patients must have close and reliable follow-up to be considered for discharge, as a thorough workup should be completed as soon as possible, preferably within 48 hours.

## KEY POINTS

- TIA is characterized by transient ischemia in the CNS that does not result in infarction (stroke) but portends an increased risk of stroke in the ensuing days and weeks.
- The ABCD<sup>2</sup> score is a tool with reasonable predictive accuracy that can be used to help risk stratify patients with TIA. It is by no means infallible and should be used alongside clinical judgment.
- The ability to complete the outpatient TIA workup must also be considered. Inpatient admission is strongly advised for moderate and high-risk patients.

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## THE ELUSIVE BRAIN ABSCESS

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**SHIKHA KAPIL, MD AND JEFFREY N. SIEGELMAN, MD**

Brain abscesses are focal or multifocal lesions within the brain parenchyma that result from direct or hematogenous spread of infection. Although the incidence is very low (ranging from 0.4 to 0.9 cases per 100,000), the mortality remains high at ~10%. Brain abscesses can be notoriously difficult to diagnose with the “classic triad” of fever, focal neurologic deficit, and headache present in only 20% of cases.

Approximately half of all cases of brain abscess occur in patients with predisposing conditions such as immunosuppression (e.g., HIV or immunosuppressive drug therapy) or exposure of the central nervous system to outside pathogens (e.g., trauma, sinusitis/mastoiditis, dental infections, or surgical intervention). Direct spread generally results in focal abscesses, while hematogenous spread results in multifocal abscesses, most frequently in the distribution of the middle cerebral artery territory.

The most common presenting symptom is headache, which is one of the most common chief complaints evaluated in the emergency department (ED). The pain associated with brain abscess is generally described by patients as severe and is not typically relieved by oral analgesics. Focal neurologic deficits occur in 50% of patients, and seizure occurs in 25% of cases. Of note, nearly half of these patients are afebrile. If the patient has an underlying predisposition and concerning symptoms, brain abscess should be in the differential diagnosis.

Computed tomography (CT) with contrast or magnetic resonance (MR) is the test of choice in the evaluation for brain abscesses. An MR with gadolinium contrast is the most sensitive test for detecting a developing abscess and can detect lesions earlier than contrast CT. Nonetheless, CT is clearly more practical in the ED setting, and CT is sufficient for ruling out abscesses that are imminently life threatening due to their mass effect. The

appearance of brain abscesses on CT depends on their chronicity, although the classic appearance is one of a ring-enhancing lesion.

It is important to note that lumbar puncture (LP) is contraindicated if an abscess is suspected, particularly in the setting of focal neurologic deficit. The procedure carries a significant risk of brain herniation (up to 30% in some studies). Moreover, the diagnostic yield of LP and cerebrospinal fluid examination is low for the diagnosis of brain abscess. If the differential diagnosis also includes meningitis, blood cultures may be drawn and antibiotics may be started empirically, but LP should not be performed until CT or MR confirms the absence of a space-occupying lesion.

The most common pathogens include *Staphylococcus* and *Streptococcus* species. A neurosurgeon should be consulted at the time of diagnosis as treatment often involves surgical drainage. Transfer to another facility is indicated if neurosurgical expertise is not available. Antibiotic therapy is typically prolonged (e.g., 4 to 8 weeks) and varies depending on patient factors, including underlying conditions and severity of disease; consultation with an infectious disease expert is warranted. [Table 195.1](#) summarizes empiric antibiotic therapy for brain abscesses based on the suspected source.

**TABLE 195.1 RECOMMENDED ANTIBIOTICS FOR BRAIN ABSCESS BY SOURCE**

| SOURCE                                     | ANTIBIOTIC   |
|--|--|
| Bacteremia                                 | Vancomycin or oxacillin  |
| HIV infection                              | Cefotaxime or ceftriaxone plus metronidazole, pyrimethamine, and sulfadiazine                        |
| Oral/otic/sinus                            | Metronidazole plus penicillin, ceftriaxone, or cefotaxime  |
| Penetrating trauma                         | Vancomycin plus ceftriaxone or cefotaxime. If sinuses or oral cavity is involved, add metronidazole. |
| Postoperative from neurosurgical procedure | Vancomycin PLUS ceftazidime, cefepime. Alternative is meropenem.                                     |
| Unknown source                             | Vancomycin plus ceftriaxone or cefotaxime plus metronidazole. Alternative is meropenem.              |

Adapted from Brouwer MC, Tunkel AR, McKhann GM II, et al. Brain abscess. *New Engl J Med*. 2014;371(5):447–456.

## KEY POINTS



- Brain abscesses can arise from a number of predisposing conditions and from either direct or hematogenous spread.
- Focal neurologic deficits and seizures should heighten suspicion in susceptible patients. Fever is absent in half of all cases. Diagnosis is made by either CT or MR imaging.
- Consultation with a neurosurgeon and infectious disease specialist is indicated. Antibiotic treatment is prolonged.

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## BULBAR SYMPTOMS IN THE ED: WATCH THE AIRWAY

ARIEL BOWMAN, MD

The bulbar muscles of the mouth, tongue, soft palate, pharynx, and larynx are responsible for speech, chewing, and swallowing. Dysfunction of these muscles can lead to dysarthria, dysphagia, and impaired gag and cough, all of which can increase the risk for aspiration and airway compromise. It can be easy to overlook these symptoms because of the subtleties of the associated examination findings and the seemingly subjective nature of complaints. Nonetheless, several conditions that result in bulbar symptoms can be life threatening; thus, they should be considered a possible early indicator of impending airway compromise and respiratory failure. While many conditions can result in bulbar symptoms, including stroke and amyotrophic lateral sclerosis (ALS), three key diagnoses that often confound the emergency provider are myasthenia gravis (MG), botulism, and Guillain-Barré syndrome (see [Table 196.1](#)).

**TABLE 196.1 CLINICAL FEATURES OF MYASTHENIA GRAVIS, BOTULISM AND GUILLAIN-BARRÉ SYNDROME**

|  | <b>MYASTHENIA GRAVIS</b>   | <b>BOTULISM</b>   | <b>GUILLAIN-BARRÉ</b>  |
|--|--|---|--|
| Pathophysiology                                    | Autoantibodies to postsynaptic AchRs.  | <i>Clostridium botulinum</i> toxin inhibits release of Ach at presynaptic membrane.   | Immune-mediated destruction of myelin sheath or axon.                              |
| Presentation                                       | Fluctuating symptoms, symptoms worse later in the day or with sustained activity.  | Descending, symmetrical flaccid paralysis.  | Ascending, symmetrical weakness, decreased DTRs; can present with facial weakness. |
| Associated symptoms                                | Ptosis, diplopia, proximal limb weakness, neck and respiratory muscle weakness. No pupillary involvement.                  | Nausea, vomiting, and diarrhea. Anticholinergic symptoms including pupillary involvement with mydriasis. DTRs normal or decreased.                      | Autonomic involvement with VS instability, urinary retention.                      |
| Diagnosis (in addition to history and examination) | Tensilon and ice pack tests, serologic testing, RNS, single-fiber EMG.   | Exclude other causes, stool and serum tests for toxin but not readily available and often delayed, anaerobic wound culture in suspected wound botulism. | CSF with increased protein without elevated WBCs, EMG, nerve conduction studies.   |
| Treatment  | Intubation, IVIG and plasmapheresis in crisis, long-term with cholinesterase inhibitors and immunosuppression, thymectomy. | Airway management, antitoxin, notify CDC and health department for testing of suspected food source, abx in wound botulism.                             | Intubation, IVIG, plasmapheresis.  |

MG is the most common disorder of neuromuscular transmission. It most frequently affects women in the teens to 30s and men aged 50 to 70. It is an autoimmune disorder in which autoantibodies attack the postsynaptic nicotinic acetylcholine receptor (AChR) at the neuromuscular junction (NMJ). Destruction of AChRs leads to fewer available binding sites for acetylcholine (ACh) and thus manifests as weakness or fatigability of muscles with repeated use—the hallmark of MG. Classically, patients present with ocular symptoms resulting in ptosis, weakness of extraocular movements, and diplopia, but ~15% of patients will initially present with bulbar symptoms involving oropharyngeal, palatal, and jaw muscles. Patients may complain of dysarthria or dysphagia, fatigable chewing, voice changes,

nasal regurgitation, and inability to hold the jaw shut. Any skeletal muscle can eventually be involved, including limb and respiratory muscles. Diagnosis of MG in the emergency department (ED) should focus on history and physical examination. Bedside testing with edrophonium (a reversible short-acting ACh inhibitor), the so-called Tensilon test, or the ice pack test can be performed with caution in the ED, but both are advised only for patients with ptosis, in which a response can most easily be quantified. Confirmation of the ED diagnosis with serologic testing for autoantibodies against the AChR as well as nerve stimulation tests and electromyography is typically performed in the outpatient setting. ED management of MG should focus on evaluation for myasthenic crisis and assessment of the patient's respiratory function with measurement of forced vital capacity (FVC) and negative inspiratory force (NIF). Intubation and admission for plasmapheresis and IVIG may be necessary for patients in crisis.

Botulism is caused by a neurotoxin produced by the gram-positive anaerobic spore-forming bacterium *Clostridium botulinum*. Three main forms of botulism exist: food-borne (ingestion of preformed toxin), infantile (ingestion of spores that subsequently produce toxin in the gut), and wound botulism (toxin production in a wound infected by *C. botulinum*). The disease is caused by irreversible binding of toxin to the presynaptic membrane at the NMJ, inhibiting the release of ACh, leading to muscle weakness and autonomic findings. Patients present with a descending, symmetric, flaccid paralysis that begins with cranial nerve and bulbar involvement and includes the muscles of respiration. There are no cognitive or sensory impairments. Patients can have signs consistent with an anticholinergic toxidrome including mydriasis. This finding will help distinguish botulism from MG, which spares the pupils. Food-borne botulism has an incubation period of 12 to 36 hours and may be accompanied by nausea, vomiting, and diarrhea. Patients with wound botulism typically have a history of minor trauma or of injection drug use, most commonly "skin-popping" of black tar heroin, and will have associated dermatologic findings on examination. There is a longer prodrome (days to weeks), and patients may have a concomitant fever, although this is likely secondary to associated bacterial infection of the wound. Infantile botulism (affecting infants <12 months of age), caused by ingestion of spores that are not killed in the less acidic infant gastrointestinal environment, can pose a diagnostic challenge. The classic "floppy baby" can also present with a blank facial expression, poor feeding, weak cry, and constipation. Key historical features are ingestion of honey or residence near a construction site, leading to exposure to spores from disrupted soil. Stool and serum assays for toxin should be ordered but should not delay presumptive diagnosis in suspected cases. Early

management of the patient's airway and supportive care are key. There is an antitoxin available in the United States from the Centers for Disease Control and Prevention (CDC) for children older than 1 year and for adults. There is also an intravenous (IV) botulism immunoglobulin available for infants (BabyBIG). Patients with wound botulism should receive antibiotics for the local infection, avoiding aminoglycosides, which may potentiate neuromuscular blockade. Wound debridement and updating of tetanus status are also recommended. Notification of the CDC and local public health department aids in securing these treatments and in testing of suspected sources.

Guillain-Barré syndrome (GBS) refers to a heterogeneous group of acute immune-mediated polyneuropathies. The most common presentation is an ascending symmetric muscle weakness with decreased deep tendon reflexes and variable sensory impairment. Although this is the classic presentation, multiple subtypes exist, including the Miller Fisher variant that may present with weakness involving the oropharyngeal, neck, and shoulder muscles. Furthermore, ~50% of all GBS patients will eventually have involvement of facial or bulbar muscles. Patients may report a preceding diarrheal or respiratory infection. Incidence increases with increasing age and men are more commonly affected than are women. Progression of disease symptoms occurs over days to weeks, with maximal symptom severity reached by 4 weeks into the disease course. Lumbar puncture will often reveal elevated protein in the cerebrospinal fluid (CSF) without a concomitant pleocytosis. The prevalence of this finding increases with duration of illness and will be present in over 75% of patients by the third week of illness. Electromyography (EMG) and nerve conduction studies are used for confirmation and prognostication. As with MG and botulism, the key ED management consideration in GBS is airway protection and respiratory status. Early intubation should be performed with signs of respiratory muscle compromise, and up to 30% of GBS patients will require intubation during the course of their illness. An FVC < 20 mL/kg and an NIF < 30 cm H<sub>2</sub>O are predictive of respiratory failure. Up to 20% of patients with GBS will have autonomic dysfunction/instability, and IV vasoactive medications may be necessary, but only short-acting agents should be used due to the potential for rapid fluctuations in vital signs as a result of the underlying disease process. Patients with or without respiratory failure should be admitted for plasmapheresis and/or IVIG treatment. Steroids are no longer recommended.

## KEY POINTS

- Bulbar symptoms involve the muscles of speech, chewing, and swallowing.
- Three conditions that may initially present with bulbar symptoms are MG, botulism, and GBS.
- Among other considerations, careful attention to airway protection is a common critical management priority in all of these patients. Close monitoring and early intubation for signs of respiratory compromise are essential.

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# **MULTIPLE SCLEROSIS IN THE ED: RULE OUT OTHER DIAGNOSES FIRST**

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**CHRISTOPHER BODLE, MD AND MELISSA WHITE, MD,  
MPH**

Multiple sclerosis (MS) is a chronic demyelinating disease of the central nervous system. The pathophysiology of MS is incompletely understood but thought to involve genetic predisposition, environmental factors, and possibly viral infection. The result is inflammatory demyelination of nerve fibers and subsequent slowing of nerve conduction. The incidence is three times greater in women than men and two times greater in Caucasians than African Americans. At higher latitudes, there is a higher incidence of disease and higher rate of relapse.

The overall course of disease in MS varies widely from patient to patient. Most have relapsing-remitting disease in which discrete attacks arise and then resolve. However, others have steady progression of disability or steady progression of disability with superimposed acute attacks. Patients can present with wide-ranging neurologic complaints including focal weakness, paresthesias, cerebellar dysfunction, cranial nerve findings, bowel/bladder dysfunction, or dysautonomia. Optic neuritis is the initial symptom in nearly 30% of patients diagnosed with MS. In patients with demyelination in the cervical spinal cord, neck flexion can cause radiation of sharp, shooting pain down the spine. This is known as *L'hermitte's sign* and can mimic cervical spinal stenosis or cervical disc herniation. The rate of generalized seizures in MS is the same as the general population; however, the rate of simple partial seizures is two times greater in patients with MS.

It is difficult to diagnose MS in the emergency department (ED). In order to make a diagnosis, there must be two or more progressive episodes of neurologic dysfunction separated in both space and time. A history of previous transient neurologic deficit in the past can be useful in evaluating current symptoms. Symptoms of MS can mimic other serious disease processes including malignancy, intracranial hemorrhage, ischemic stroke, lupus, Lyme disease, and neurosyphilis. Further complicating the presentation of MS, body temperature can alter the presentation of symptoms. Known as *Uhthoff's phenomenon*, current neurologic deficits in MS can be worsened by elevated body temperature, and previous neurologic deficits can present again (*recrudescence*). Thus, patients with MS and elevated temperature (from fever or the environment) can have more severe neurologic deficits than at baseline.

In patients who present to the ED with symptoms possibly consistent with MS, a workup is necessary to rule out other serious causes of neurologic dysfunction. This workup can generally be deferred to a neurologist in the outpatient setting in a timely fashion. Brain magnetic resonance (MR) will generally show acute and chronic white matter lesions. It is important to note that computed tomography (CT) cannot reliably identify MS lesions. It is nonetheless indicated as an initial study in the ED to rule out other causes of neurologic deficit. Cerebrospinal fluid analysis (CSF) can demonstrate elevated protein, but not always. Moreover, elevated CSF protein is neither sensitive nor specific for MS; it can also be elevated in bacterial meningitis or subarachnoid hemorrhage or with a traumatic tap. CSF analysis for *oligoclonal bands* is used in the diagnosis of MS but outside of the scope of the ED workup.

MS is treated with immunosuppressive medications. There are two arms of treatment. Long-term treatment with chronic disease-modifying therapy can slow the progression of disease and decrease the frequency of acute attacks. In acute attacks, glucocorticoid therapy has been shown to be of benefit by decreasing the severity and length of the attack. Intravenous glucocorticoids are generally initiated for any MS patient being admitted to the hospital with an acute attack. Early consultation with a neurologist is recommended to guide early treatment and disposition in patients with concerning symptoms.

## KEY POINTS

- Patients with MS can present with wide-ranging neurologic



complaints including focal weakness, paresthesias, cerebellar dysfunction, cranial nerve findings, bowel/bladder dysfunction, and dysautonomia.

- ED evaluation centers on ruling out other life-threatening causes of symptoms and may include CT imaging and CSF examination.
- Consultation with a neurologist is recommended to guide early treatment and disposition in patients with concerning or rapidly evolving symptoms.

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# **SECTION XIV**

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## **OB/GYN**

# EARLY PREGNANCY: SIFTING OUT THE POTENTIAL CATASTROPHES FROM THE WORRIED WELL

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JARED T. MARX, MD, FACEP

A patient presenting to the emergency department (ED) in early pregnancy may represent a patient who is a potential catastrophe or simply the “worried well.” Despite technologic advancements, diagnosing ectopic pregnancy is not always straightforward in the ED since patients may present with a wide array of complaints and may not even realize they are pregnant upon presentation, and the gold standard remains diagnostic laparoscopy.

Every woman of reproductive age presenting to the ED should have a pregnancy test performed. A positive test may alter differential diagnoses, imaging selection, or medications prescribed.

Pregnancy is often tested by a point of care urine  $\beta$ -HCG test. A negative result can be taken with a grain of salt. These tests are performed by busy personnel pulled in many directions. Distraction may lead to a premature interpretation leading to a false-negative result. When there is high suspicion that a complaint is pregnancy related, consider a serum  $\beta$ -HCG.

Vital signs are essential to monitor and trend in pregnant patients. Any pregnant patient with critical vital signs requires an emergent OB consultation without delay while performing or awaiting other test results.

Ultrasound is the go-to in the ED when risk stratifying a patient for ectopic pregnancy, whether performed at bedside or in radiology. In any female patient who is **NOT** undergoing treatment with reproductive assistance, the identification of an intrauterine pregnancy (IUP) essentially rules out an ectopic pregnancy.

Simply placing an ultrasound probe on the patient's abdomen and identifying a pregnancy is not sufficient to differentiate IUP versus ectopic pregnancy. Uterine anatomy should be well defined with meticulous ultrasound imaging of the uterus in both long and short axes. To determine a pregnancy as intrauterine, it should be identified within the endometrial canal of the uterine fundus in both planes.<sup>1</sup>

The pelvis should be evaluated for a significant amount of free fluid, defined as free fluid extending  $>1/3$  the posterior wall of the uterus or extending outside the pelvis. In addition, bilateral adnexa should be evaluated for masses. Either finding may suggest an ectopic pregnancy.

Identification of an anechoic (black) structure in the endometrial canal of the uterine fundus may not always represent an IUP. A pseudogestational sac may be present from the endometrial decidual reaction despite the patient having an ectopic pregnancy. A definitive IUP can be identified if the gestational sac is located in the fundal endometrial canal and it contains an identifiable yolk sac. Although a double decidual sign is arguably an earlier sign of IUP, it is only present in about 50% of pregnancies and may be mistakenly identified as present when not.

If an IUP cannot be identified by transabdominal ultrasound then transvaginal ultrasound should be performed. Transvaginal ultrasound allows for identification of pregnancy structures ~1 week earlier than transabdominally. It also improves the ability to evaluate for pelvic free fluid and to identify adnexal masses.

Blood work has little value in the early evaluation of pregnancy if an IUP can be definitively diagnosed, and can be limited to specific situations.

Rh typing: If unknown, patients should undergo blood typing. Bleeding patients found to be Rh negative should receive Rho (D) immune globulin to prevent future pregnancy complications.

Quantitative serum  $\beta$ -HCG: This test has little value if a definitive IUP (yolk sac present within the gestational sac) has been identified by ultrasound. A quantitative  $\beta$ -HCG discriminatory zone level may be helpful when pregnancy location cannot be identified by transvaginal ultrasound. The discriminatory zone is the quantitative  $\beta$ -HCG level above which the early signs of an IUP should be identifiable by ultrasound. It may vary by institution but usually lies between 1,000 and 2,000.<sup>2</sup>

The discriminatory zone should be approached with caution. An empty uterus with a  $\beta$ -HCG above the discriminatory zone represents ectopic pregnancy until proven otherwise, and OB should be consulted to the ED.

Although a level below the discriminatory zone may indicate an early

pregnancy or miscarriage, it may also represent ectopic pregnancy. Up to 40% of ectopic pregnancies have serum  $\beta$ -HCG levels below the discriminatory zone upon presentation.<sup>2</sup>

A patient with an empty uterus and  $\beta$ -HCG levels below the discriminatory zone, stable vital signs, no adnexal masses, and only small amounts of free fluid in the pelvis requires close follow-up established with OB. These patients need repeat  $\beta$ -HCG levels and ultrasound to identify pregnancy presence and location.

A patient with an empty uterus with  $\beta$ -HCG levels below the discriminatory zone, abnormal vital signs, an adnexal mass, **OR** significant free fluid in the pelvis requires OB consultation for suspected ectopic pregnancy.<sup>3</sup>

CBC and CMP: These values offer little assistance in the care of pregnant patients unless there is significant bleeding, an ectopic pregnancy is identified or suspected, or the patient's complaints otherwise indicate their usage. Both are valuable and will need trended when the treatment plan for ectopic pregnancy is medical management with methotrexate.

Urine and vaginal infections raise the risk of miscarriage and should be treated, including asymptomatic bacteriuria. Urine samples and vaginal and cervical swabs should be obtained in all pregnancy-related complaints.

Sifting the potential catastrophes from the “worried well” in early pregnancy is not always straightforward, but meticulous examination of the pelvic structures by ultrasound with ancillary labs when needed is essential for safe effective care in this population.

## KEY POINTS

- Meticulous evaluation of the uterus in long and short axis is key to identifying pregnancy location.
- Definitive IUP is a gestational sac containing a yolk sac lying within the fundal endometrium.
- $\beta$ -HCG values can be helpful when the uterus is empty on ultrasound, but levels below the discriminatory zone may still represent ectopic pregnancy and close follow-up should be established.

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# **PITFALLS IN THE PURSUIT OF OVARIAN TORSION**

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While the overall incidence of ovarian torsion is fairly low, cases of ovarian torsion that present to the emergency department have a high rate of misdiagnosis on the initial visit, have significant associated morbidity, and can be a source of significant medicolegal risk to the provider. Ovarian torsion should therefore be a part of the differential diagnosis when evaluating any female with abdominal pain. Being aware of several persistent myths and misconceptions that exist may allow providers to refine their workups and more accurately estimate pre- and posttest probability of torsion.

## **CLASSIC DOESN'T MEAN COMMON**

Classically, pain from ovarian torsion has been described as sharp, sudden, unilateral pain occurring in women of reproductive age. In practice, the patient's demographics and symptoms have significant variability; however, several features may increase the pretest probability of ovarian torsion. In a retrospective review, 70% of patients reported "sharp or stabbing" pain, symptoms had an abrupt onset in only 59% of cases, and 70% of patients reported associated nausea and vomiting.<sup>1</sup> The majority of cases of torsion occur after the onset of menarche; however, up to 15% of cases occur in the pediatric population with a similar percentage of cases occurring in patients who are postmenopausal, and roughly 20% of cases of ovarian torsion occur during pregnancy.<sup>2</sup> While most patients with ovarian torsion have a structurally abnormal ovary, patients commonly have no preceding history with only 25% of patients reporting a previous history of ovarian cyst or

mass. In pediatric cases, up to 58% of patients had no obvious ovarian pathology. Features such as a history of previous pelvic surgery or pelvic inflammatory disease may increase the likelihood of a patient developing torsion; however, these historical elements are present in a minority of the reported cases.

## **DON'T RELY ON YOUR BEDSIDE EXAM**

Bedside exam is rarely helpful when trying to make the diagnosis of ovarian torsion. The presence of abdominal pain, pelvic mass, or significant adnexal tenderness may increase the likelihood of ovarian torsion, yet in practice, the bedside exam suffers from poor sensitivity and specificity and should not be used to rule out cases of torsion. In addition, the sensitivity of the pelvic exam for detecting adnexal masses ranged from 15% to 36% despite being performed under near ideal circumstances by gynecology attendings in an operative setting.<sup>3</sup>

## **CT MAY BE AN ADEQUATE STUDY**

For patients with a convincing story for possible torsion, ultrasound is the most reasonable first-line imaging study. Given the variable presentations of torsion, providers may instead order a CT of the abdomen/pelvis when patients present with a complaint of nonspecific abdominal pain. When faced with a nondiagnostic CT scan, providers will order an ultrasound to more fully evaluate for ovarian pathology including torsion. Recent studies have suggested that CT may be a reasonable imaging modality and have questioned the added utility of ultrasound with reported sensitivities and specificities of 80% to 100% for both CT and US when diagnosing ovarian torsion.<sup>4</sup> Given this diagnostic accuracy, when torsion is seen on the CT scan, patients do not need further imaging.

More commonly, providers have a patient with ongoing pain and a CT scan that does not show torsion. In this scenario, CT scan may be helpful in terms of identifying other ovarian pathology that could lead to the patient developing torsion. The incidence of ovarian torsion occurring in patients with completely normal imaging is extremely low. When compared to ultrasound, CT has been shown to be more likely to detect abnormal ovarian pathology. Given the high sensitivity of CT scan for detecting ovarian pathology and the rare incidence of torsion in patients with normal ovarian anatomy, a truly negative CT scan in a patient with abdominal pain may effectively rule out ovarian torsion in a large majority of patients.



# ULTRASOUND HAS SIGNIFICANT LIMITATIONS

Ultrasound has a wide range of reported sensitivities (~36% to 85%) in the evaluation of patients with potential ovarian torsion. When present, features such as a lack of blood flow or obvious ovarian edema have a high degree of diagnostic accuracy. While Doppler ultrasound can be diagnostic when used to identify a lack of blood flow, normal Doppler scans are seen in up to 1/3 of patients with surgically proven ovarian torsion.<sup>5</sup> As with CT scans, cases of torsion in a patient with normal ovarian anatomy on ultrasound do occur, but are exceedingly rare. MRI has good reported diagnostic characteristics; however, logistically, it may be difficult to obtain in a timely fashion when evaluating a case of acute abdominal pain.

Given the limitations associated with patient history, exam, and imaging, providers should remain vigilant for patients who present with a concerning story for ovarian torsion. In any scenario where the patient has a high pretest probability of ovarian torsion, providers should have a low threshold to consult OB/GYN despite negative imaging studies if there is ongoing clinical concern for ovarian torsion.

## KEY POINTS

- History and exam cannot be used to reliably rule out ovarian torsion.
- CT scan can reliably identify torsion in the majority of cases.
- Ultrasound may be normal in the setting of intermittent torsion.
- Torsion is exceedingly rare in patients who have completely normal ovarian anatomy.
- If concerned for intermittent torsion, providers should consult OB/GYN.

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## **ANTI-D IN THE ED**

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A small minority of patients does not express the D rhesus surface antigen on erythrocytes. These patients are designated Rh negative. About 15% of the Caucasian population is Rh negative, and the prevalence is much lower in other ethnic groups; about 5% to 8% in African Americans and only 1% to 2% in Asians and Native Americans. If an Rh-negative mother has an Rh-positive fetus, fetomaternal hemorrhage can result in fetal blood entering the maternal circulation. This causes the mother to produce anti-D antibodies in response to the perceived antigen. This process is termed sensitization or alloimmunization. While this does not usually result in any immediate adverse effects, as IgM antibodies do not cross the placenta, it can cause significant problems in subsequent pregnancies. If a sensitized mother is exposed to Rh-positive erythrocytes in a future pregnancy, IgG antibodies are rapidly produced that can cross the placenta and result in fetal anemia, hemolytic disease of the newborn, and intrauterine fetal death.

The development of anti-D immune globulin (commonly referred to by the trade name RhoGAM in the United States) has dramatically reduced the rate of maternal sensitization. The mechanism by which anti-D works is still unproven. However, combined antepartum and postpartum administration per established protocols clearly decreases the incidence of alloimmunization. Clear guidelines exist for the administration of anti-D to Rh-negative women in the third trimester although specific dosing regimens vary by country. Of course, this is outside the scope of the emergency physician. However, the emergency physician will regularly take care of pregnant patients who have experienced potentially sensitizing events. Knowing what these events are, the indications and dosing for anti-D, and where the evidence is lacking will allow you to provide effective and efficient care for your patients.

Considerable controversy remains regarding the administration of anti-D in the first trimester in cases of threatened miscarriage. Current practice in the United States is largely based on a 1999 American College of Obstetricians and Gynecologists practice bulletin, which admits that their consensus is based in expert opinion and “no evidence-based recommendation can be made.”

So what does the evidence show? First, the fetal red blood cells begin expressing the Rh(D) antigen as early as 38 days after conception, or 52 days from the last menstrual period. So before this time period, there is not even a theoretical risk of sensitization. As a pregnancy progresses, there is a theoretical risk of sensitization due to threatened miscarriage. However, there are no documented cases in the literature of this occurring in the first 12 weeks of gestation without some other complicating factor. The only randomized controlled trial attempting to study this showed no cases of alloimmunization in either the treatment or placebo group. There is still no clear evidence to guide decision-making in this context. Some have argued that since the safety profile of anti-D is good and that side effects are rare, we should err on the side of administering it. Others point out that anti-D is difficult to produce and that resources should not be allocated to a treatment with no demonstrable benefit. Despite the ongoing controversy, what does seem clear is that anti-D is not indicated prior to 52 days from the last menstrual period or in cases of mild spotting. Unfortunately, this appears to be a scenario in which medical dogma, and not evidence, guides prevailing practice patterns.

The other major indication for anti-D administration in the emergency department setting is trauma, particularly blunt abdominal trauma. Because these patients may present without any vaginal bleeding, the risk of sensitization is often over looked by emergency physicians. Particularly if trauma occurs prior to the routine administration of anti-D at 28 weeks to Rh-negative women, the risk of sensitization is significantly increased. Fetomaternal hemorrhage has been estimated to occur in 25% to 30% of pregnant trauma patients. Greater volume hemorrhage does increase the risk of sensitization; however, the severity of trauma does not necessarily correlate with the risk of hemorrhage. Even a small amount of hemorrhage can result in sensitization. Therefore, all pregnant trauma patients should have their Rh status evaluated. Those who are Rh negative should receive 300 µg of anti-D immunoglobulin. This dose is thought to be sufficient for up to 30 mL of fetomaternal hemorrhage. If there is concern for larger volume of hemorrhage, OB should be consulted to discuss further testing such as a Rosette test and/or Kleihauer-Betke test.

Finally, in all cases where fetomaternal hemorrhage is of concern,

consider utilizing the electronic health record as a tool to determine a patient's Rh status. Waiting for an Rh determination often results in significantly increased length of stay. All pregnant women will have their type determined early in pregnancy as part of routine care. Women may also have had a prior visit to the emergency department that included determination of Rh type. Quickly reviewing old records before reflexively ordering this test on any patient with a potentially sensitizing event can reduce costs and improve departmental efficiency.

## KEY POINTS

- Patients with early first trimester threatened miscarriage (before 52 days) and minor bleeding do not need anti-D.
- Consider any significant trauma to be a potentially sensitizing event and determine the patient's Rh status.
- Use the electronic health record to confirm Rh status and avoid unnecessary and time-consuming testing.

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# SEIZING YOUNG WOMAN? THINK ECLAMPSIA. THINKING ECLAMPSIA? THINK AGAIN

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**KENNETH D. MARSHALL, MD, MA**

Actively seizing or postictal patients are a common presentation in the emergency department (ED). One cause in particular is worth special consideration, since its pathophysiology and treatment are unique, diagnostic clues sometimes surprisingly subtle, and mismanagement may lead to greater morbidity or even death. Eclampsia must be a primary consideration in all seizing women of childbearing age.

Estimates of eclampsia incidence range from 1 in 1,000 to 1 in 5,000.<sup>1-3</sup> It is classically defined as new-onset seizures in a pregnant woman with clinical features of preeclampsia, not clearly attributable to other causes. However, presentations may vary widely from this paradigm. Special attention is drawn to three particular variations from the norm, to which the emergency physician (EP) must be vigilant to correctly recognize, diagnose, and treat eclampsia.

The first variation is the pregnant woman with first-time seizures without typical features of preeclampsia, in particular, lacking proteinuria or hypertension. This occurs in 15% to 40% of eclamptic patients.<sup>3,4</sup> Subsequent investigation often reveals signs or symptoms of preeclampsia (headache, visual changes, right upper quadrant pain) antecedent to the seizure. However, up to 25% of eclamptic patients present without premonitory signs or symptoms.<sup>5</sup> Therefore, absence of the hallmark features of preeclampsia should not offer the ED physician reassurance, nor deter workup (or even initiating treatment) for eclampsia.

The second variation is postpartum eclampsia. The majority of eclampsias occur ante- or intrapartum, but 20% to 25% occur postpartum. Most occur within 48 hours of delivery, but may occur throughout the 6-week puerperium.<sup>6</sup> Despite effective management reducing the incidence of ante- and intrapartum eclampsia, the relative incidence of postpartum eclampsia has risen.<sup>7</sup> Therefore, it is very possible for a seizing or postictal nongravid female, unable to provide history of her recent delivery, to present manifesting eclampsia.

The third variation is that of eclampsia in unsuspected pregnancy. Preeclampsia and eclampsia can provide the initial symptoms and signs of previously undiagnosed late pregnancy.<sup>8,9</sup> Molar pregnancies may even produce severe preeclampsia and eclampsia as early as 15 to 20 weeks of gestational age.<sup>10</sup>

There is a critical counterpoint to the above discussion. While eclampsia can be an elusive diagnosis, it also remains a diagnosis of exclusion. Even when eclampsia is a likely explanation for a seizing patient, failure to exclude other causes is folly. Ischemic or hemorrhagic strokes, as well as cerebral venous sinus thrombosis and arterial dissection, can produce seizures and altered sensorium. To make it even more complex, strokes may be caused by eclampsia. TTP-HUS, another potentially grave condition precipitated by pregnancy, may also cause convulsions and altered mental status, and its defining laboratory features can mimic the HELLP syndrome of severe preeclampsia.

A structured approach to the diagnosis of new-onset seizures in a young woman should be undertaken. Diagnostic laboratory testing generally includes a complete blood count, renal and hepatic profiles, coagulation profile, toxicology screen, and beta human chorionic gonadotropin ( $\beta$ -HCG) level. If the  $\beta$ -HCG is positive, an LDH, a magnesium ( $Mg^{2+}$ ) level, and a urinalysis for protein should be obtained as well. Advanced imaging includes a noncontrast CT of the head.

If the seizing patient is pregnant with features typical of uncomplicated eclampsia (hypertensive, laboratory features of preeclampsia, return to baseline mental status after brief seizure), treatment for eclampsia should be initiated and no further diagnostic testing is generally necessary. However, if eclampsia is considered but the presentation is atypical (postpartum, focal neurologic deficits, persistent visual disturbances, symptoms refractory to  $Mg^{2+}$ , and antihypertensives), expanded diagnostic testing, including MRI/MRA, is recommended.<sup>11</sup>

Management goals of eclampsia are to prevent secondary injury from

seizures (hypoxia, trauma), prevent recurrent seizures, control marked hypertension, and arrange prompt evaluation for delivery. Delivery is the definitive treatment for preeclampsia and eclampsia. EPs need to remain aware of these many and varied presentations of eclampsia, standing ready to diagnose and manage them correctly.

## KEY POINTS

- Eclampsia can occur without preceding symptoms.
- Eclampsia can occur without hypertension or proteinuria.
- Eclampsia can occur up to several weeks postpartum.
- Eclampsia that is atypical (focal neurologic deficits, persistent visual changes, seizures, or altered mental status refractory to treatment) needs comprehensive neurologic workup, likely including MRI/MRA.

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# VAGINAL BLEEDING IN LATE PREGNANCY

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**HEATHER A. HEATON, MD**

The role of the emergency physician in late pregnancy vaginal bleeding is stabilization of the mother and fetus with identification of maternal and fetal life-threatening etiologies after resuscitation, including intravenous fluids, monitoring, blood products when necessary, and delivery if appropriate. Decelerations or loss of variability seen with continuous fetal monitoring may resolve with maternal resuscitation; however, if fetal monitoring is persistently nonreassuring, the patient may need urgent cesarean delivery.

Initial assessment by the emergency provider should include a history, a physical exam with vital signs, and an ultrasound for evaluation of placental location. A sterile vaginal speculum exam can be performed to quantify the amount of bleeding and determine an etiology. The digital cervical exam should not be performed until placental location is known.

This chapter provides an overview to the most common, emergent etiologies of vaginal bleeding in pregnancies >20 weeks. Not to be confused with “bloody show,” a term used to describe the small amount of blood with mucus discharge that may precede labor by as much as 72 hours, placenta previa, placental abruption, uterine rupture, and vasa previa are discussed.

## PLACENTA PREVIA

Classically taught as vaginal bleeding in women >20 weeks pregnant that is painless, placenta previa is defined as placental implantation that overlies or is within 2 cm of the internal cervical os.

## ***Clinical Course***

It is often diagnosed during early second trimester anatomy scans, and patients are typically asymptomatic. Approximately 90% of the early gestation cases resolve, though in advanced gestational age resolution is less likely. In 70% to 80% of placenta previa cases, painless vaginal bleeding presents in the second half of pregnancy. However, 10% to 20% have associated uterine contractions.

## ***Diagnosis***

Caregivers should consider placenta previa as an etiology for vaginal bleeding in any woman beyond 20 weeks' gestation. Diagnosis is made by identification of placental tissue covering the internal cervical os by ultrasound.

## ***Management***

Placental previa increases risk of antepartum, intrapartum, and postpartum hemorrhage. Most neonatal morbidity and mortality results from prematurity, and therefore, the primary management strategy is prolonging pregnancy until fetal lung maturity is reached. Ultimately, disposition from the emergency department (ED) typically follows a discussion with the obstetrical team.

# **PLACENTAL ABRUPTION**

In contrast to the typical painless vaginal bleeding of placental previa, placental abruption is often described as painful. Placental abruption is the separation of the placenta from the uterine wall before delivery. It is considered the most common cause of serious vaginal bleeding, occurring in 1% of pregnancies with significant neonatal mortality. Neonatal death occurs in 10% to 30% of cases.

## ***Clinical Course***

Frequently, pregnant women present with vaginal bleeding, uterine tenderness or back pain, and evidence of fetal distress. In 10% of abruptions, disseminated intravascular coagulation (DIC) can result from the release of thromboplastin into the maternal circulation with the placental separation.

## ***Diagnosis***

Although ultrasound normally allows providers to ascertain the etiology of vaginal bleeding, it is not a reliable means for diagnosing placental abruption. Failure to identify abruption on ultrasound should not delay management.

## ***Management***

Given the significant morbidity and mortality associated with abruption, aggressive resuscitation is recommended with continuous fetal monitoring to assess fetal well-being. If fetal heart tracing is not reassuring, activate hospital resources for rapid delivery, usually cesarean, as perinatal death can occur within hours of admission.

## **UTERINE RUPTURE**

Uterine rupture can be a difficult diagnosis to make in the laboring patient. If an epidural has been placed, women are often unable to describe the pain. For those presenting to the ED, consider uterine rupture if palpation of the uterus seems odd in shape.

## ***Clinical Course***

During initial assessment of the gravid female patient at >20 weeks' gestation presenting to the ED, fetal assessment is key. Fetal bradycardia is the most common symptom associated with uterine rupture, but it is not sensitive or specific. Abdominal pain and signs and symptoms consistent with intra-abdominal hemorrhage including hypotension and tachycardia are common maternal presenting symptoms. Vaginal bleeding is not always observed with this diagnosis. Additionally, providers might observe uterine tenderness, change in uterine shape, loss of station of the fetal presenting part, or cessation of uterine contractions in these patients.

## ***Management***

Management in the ED includes stabilization as best as possible. Immediate consultation of the obstetrical service is imperative for cesarean and repair of the rupture site or hysterectomy. Maternal and fetal outcome both depend on the location and size of the rupture, as well as the speed of the intervention.

## **VASA PREVIA**

Although uncommon, vasa previa has a significant rate of perinatal mortality,

ranging from 33% to 100%. Vasa previa results in fetal vessels between the cervix and the fetal presenting part.

## ***Clinical Course and Presentation***

In patients presenting to the ED with vaginal bleeding associated with spontaneous ruptures of membranes, consider vasa previa as the diagnosis. Of note, blood is fetal in origin. Exsanguination can occur quickly given the average term fetal blood volume is ~250 mL.

## ***Management***

Management should not be delayed while the provider tries to determine if the blood is maternal or fetal. Maternal hemodynamic instability or nonreassuring fetal heart tones should prompt quick discussion and decisions with the obstetrical team regarding delivery. High-risk women are screened and usually undergo scheduled cesarean delivery to prevent complications associated with this type of umbilical cord insertion.

### **KEY POINTS**

- The digital cervical examination should not be done in pregnant women with vaginal bleeding until the placenta location is known.
- Placenta previa typically resolves during the gestational period; however, painless vaginal bleeding is a concerning presenting complaint in a pregnant female.
- Placental abruption requires quick diagnosis as associated maternal and fetal morbidity and mortality are high.
- Consider uterine rupture as an etiology of hemorrhage shock in the pregnant patient presenting after trauma, regardless of presence or absence of vaginal bleeding.
- Vasa previa is associated with fetal bleeding; fetal demise is common if bleeding is associated with this condition.

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# PREDICT THE UNPREDICTABLE: PRETERM LABOR

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**PRIYA KUPPUSAMY, MD**

There are quite a few obstetric emergencies that are known to induce tachycardia, diaphoresis, and profound incontinence in even the most competent physician. Preterm labor leading to precipitous delivery is certainly on that list. No one wants to be the physician who discharges a patient home with the diagnosis of Braxton Hicks contractions when she happens to be evolving into preterm labor. As an emergency physician, you are not expected to be Miss Cleo the psychic, but you have to predict the unpredictable when it comes to preterm labor! Luckily, you will have more than just a crystal ball in your diagnostic toolbox to help you out.

Every year, 15 million babies are born preterm and ~1.1 million die of prematurity-related complications, making preterm labor a major cause of perinatal morbidity and mortality.<sup>1</sup> Prediction of spontaneous preterm birth is poor, which makes it difficult to target interventions appropriately. Preterm labor is defined as onset of strong contractions of the uterus lasting >30 seconds and occurring at least four times every 20 minutes, resulting in changes of the cervix (effacement of at least 80% and dilation >2 cm) before 37 weeks of gestation.<sup>2</sup> Braxton Hicks contractions, on the other hand, are characterized by irregular contractions without any associated cervical changes. They are typically painless or of mild intensity but can progress to true labor.

Risk factors associated with preterm labor include previous history of preterm labor, multiparous pregnancy, cervical dysfunction, uterine abnormalities, low pregnancy weight, infection, prepartum bleeding, and preterm rupture of membranes; however, more than 50% of women who go

into preterm labor have no known risk factors.

The diagnosis of true labor can be quite difficult. Symptoms can vary from menstrual cycle cramps to isolated back pain. Useful emergency department tests include CBC, urinalysis, and pelvic ultrasound. Pelvic ultrasound can reveal a shortened cervix, which places patients at higher risk for preterm delivery. Vaginal progesterone promotes “uterine quiescence” and is often administered to asymptomatic patients with shortened cervix ( $\leq 20$  to 25 mm) in order to prevent preterm labor.<sup>3</sup> Studies have shown that application of vaginal progesterone can reduce the rate of preterm birth at  $<33$  weeks by 45% as well as decrease the rate of neonatal complications.<sup>3</sup> Cervical cerclage placement is often considered to be a method of prevention in patients with history of preterm delivery and shortened cervix, though vaginal progesterone is just as effective and does not require anesthesia or surgery.<sup>3</sup>

Fetal fibronectin has emerged as a useful tool to predict preterm labor in both symptomatic and high-risk asymptomatic women. It is a glycoprotein found in amniotic fluid and placenta. Detectable during the first 22 weeks of pregnancy, fetal fibronectin reflects normal growth and function. It is usually absent from the cervical vaginal fluid starting at the 24th gestational week until near term, after which it becomes detectable again. It is released after mechanical or inflammatory-mediated damage to the placenta or membranes before birth. When present in high concentrations within cervicovaginal fluid, this protein signals increased risk of preterm labor in asymptomatic and symptomatic women. The test has a high negative predictive value. When tested between 24 and 34 weeks of gestation, a negative result ( $<50$  ng/mL) means that there is little possibility of preterm delivery within the next 7 days.<sup>4</sup> Initially, this was only available as a qualitative test. A rapid, bedside quantitative measure of fetal fibronectin level has emerged as a way to enhance the positive predictive value. A recent prospective masked observational cohort study of cervicovaginal fluid quantitative fetal fibronectin concentration in asymptomatic women found that 9.5% of women delivered preterm with values  $<10$  ng/mL, whereas 55.1% delivered with values  $>200$  ng/mL.<sup>5</sup>

The fetal fibronectin test is easily performed. During speculum exam, a swab specimen is collected from the posterior fornix of the cervix. Leave the swab in the posterior fornix for at least 10 seconds prior to removal. Most labs can turn over results within an hour. Keep in mind that a false-positive fetal fibronectin result can occur if the test is performed after bimanual examination of the cervix or after sexual intercourse up to  $\sim 24$  hours prior. The presence of vaginal bleeding and use of lubricant can also skew the



results.<sup>6</sup>

Once preterm labor is diagnosed, there are several interventions that should be considered in conjunction with close OB/GYN involvement. Place patients in Trendelenburg position, where the stretcher is adjusted so that the patient's feet are higher than her head by 15 to 30 degrees, and place a bump (rolled towel, etc.) under the right hip to shift the gravid abdomen to the left and off of the mother's inferior vena cava. Tocolytic agents can delay preterm delivery for at least 48 hours, which can allow time for administration of corticosteroids for fetal lung development. It's also important to consider antibiotics for group B strep prophylaxis.<sup>7</sup>

A sound understanding of preterm labor is quintessential for the emergency physician, since early detection and prediction is difficult. Symptoms can be mild and easily slip under the radar during the initial stages. In the later stages, however, it is often too late.

## KEY POINTS

- Ultrasound can evaluate for shortened cervical length, which places pregnant women at higher risk of preterm labor.
- Fetal fibronectin testing (both qualitative and quantitative) is an effective way to rule out preterm labor, given its high negative predictive value.
- A false-positive fetal fibronectin result can occur if the test is performed within 24 hours of sexual intercourse or bimanual exam.
- Once diagnosis of preterm labor is made, consider tocolytics, steroids, and group B strep prophylaxis in conjunction with emergent OB/GYN consult.

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# A BUMP ON THE BUMP: MINOR ABDOMINAL TRAUMA IN PREGNANCY

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Trauma is the most common cause of nonobstetrical death in pregnant women,<sup>1</sup> and the risk of domestic abuse significantly increases during pregnancy.<sup>1,2</sup> Emphasis on systematic evaluation is critical to successful emergency department management of pregnant trauma patients and crucial in providing the best likelihood of survival to both the mother and fetus.

## PRIMARY AND SECONDARY SURVEY

As with the nonpregnant patient, begin with the primary survey—**AIRWAY, BREATHING, and CIRCULATION**<sup>3</sup>—but keep in mind the physiologic changes brought on by pregnancy. Pregnant patients will have a heavy, bulky mass in the abdomen resulting in decreased tidal volume and a baseline tachypnea.<sup>3</sup> Increased pressure on the inferior vena cava (IVC) will decrease venous return to the heart and predispose the patient to hypotension.<sup>1,3</sup> Due to changes in circulating volume and systemic vascular resistance, pregnant patients can lose a significant amount of blood before tachycardia or hypotension develops.<sup>1,3</sup>

The first and easiest resuscitative measure is positioning. Placing the pregnant patient in reverse Trendelenburg with even mild left lateral decubitus positioning can remove the pressure on the diaphragm and allow easier breathing or ventilatory assistance via bag-valve mask and relieve the

pressure of the fetus from the IVC to increase preload to the heart.<sup>1,3</sup> If the patient is still on full spine precautions, place a folded blanket under the right side of a backboard for the same effect.<sup>4</sup>

Minor and major trauma in the pregnant patient is managed in much the same way as is that in nonpregnant patients, though it is important to note that priority must be given to evaluation and treatment of the *mother* rather than the fetus.<sup>4</sup> If vaginal bleeding is present in a patient who is in her third trimester, disruption of the placenta may have occurred.<sup>1,3</sup> Evaluation of the perineum and vagina should be performed to evaluate for cervical effacement, dilation, fetal presentation, presence of amniotic fluid, or blood.<sup>1</sup> Laceration from an “open” pelvic fracture may also be noted.

## LABORATORY CONSIDERATIONS IN PREGNANCY

The most critical laboratory test in the pregnant patient is the type and screen. Fetomaternal hemorrhage can occur in up to 40% of all pregnant trauma patients.<sup>1,4</sup> Women with Rh-negative blood are at risk for developing immune sensitivity, which can put all future pregnancies at risk. The degree of hemorrhage can be quantified with a Kleihauer-Betke test (which stains fetal RBCs in maternal blood, but not maternal cells), though there is risk of sensitization even with a negative KB test.<sup>4</sup> Therefore, consider giving RhoGAM to all Rh-negative patients.<sup>1</sup>

## IMAGING

Radiation is another area of concern when caring for the pregnant trauma patient. In the case of suspected serious injury, it is never appropriate to withhold critical diagnostic imaging for the sake of the fetus.<sup>3</sup> When applicable, it is appropriate to shield the belly during imaging or to choose an alternative study such as MRI or ultrasound.<sup>4</sup> While many patients may be resistant to the idea of exposing their fetus to the harmful effects of radiation, it is important to emphasize the fact that there is unlikely to be any significant effect of diagnostic radiation exposure after 15 weeks' gestation.<sup>1</sup>

## PLACENTAL ABRUPTION/UTERINE RUPTURE

Several pregnancy-related traumatic injuries include placental abruption and uterine rupture. Both conditions carry a high risk of fetal demise as high as 50% in some studies of placental abruption and should be suspected in the

presence of abdominal pain, vaginal bleeding, signs of shock, or nonreassuring fetal heart rate.<sup>3,4</sup> Abruptio remains a clinical diagnosis but can be supported by the presence of abnormal coagulation labs (platelets, fibrinogen) and/or the presence of a subchorionic hemorrhage, which increases risk.

## FAST EXAM

In the pregnant patient with minor abdominal trauma and no initial signs of placental abruption or uterine rupture, an appropriate workup is a thorough history and physical exam, followed by bedside ultrasonography. This sonographic evaluation is used to rule out significant intra-abdominal trauma (FAST exam to evaluate for free intraperitoneal fluid) and to evaluate the condition of the fetus.<sup>1</sup> Worrisome signs include abnormal fetal HR (normal range = 120 to 160), lack of fetal heart tones or fetal movement, hemorrhage into the amniotic cavity, or subchorionic hemorrhage, all of which confer a high risk of fetal demise.

## FETAL MONITORING IN TRAUMA

In the absence of any initial findings of serious maternal trauma or fetal demise, all viable fetuses (generally considered >23 weeks) should be monitored for at least 4 hours.<sup>1,2</sup> This is generally performed in an L&D triage setting due to the need for continuous fetal monitoring and tocometry. Abnormalities during this time may indicate the need for further monitoring or intervention, but if there are no signs of fetal distress, persistent uterine contractions, or rupture of membranes during this 4-hour period, patients should generally be considered safe for discharge.<sup>4</sup>

### KEY POINTS

- Domestic abuse increases during pregnancy: Screen for it!
- The first priority is the mother, not the baby: The best initial treatment for the fetus is providing optimal resuscitation to the mother!
- Radiation exposure is a concern in trauma, but do not withhold imaging.
- Type and screen: Give RhoGAM if Rh NEGATIVE.
- Tilt pregnant patient toward her left side.

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# **STABLE IS THE NEW ABNORMAL: BEWARE THE NORMAL VITAL SIGNS IN PREGNANCY**

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**PRIYA KUPPUSAMY, MD**

Vitals signs in pregnancy are exactly that: VITAL. They serve as a window into the overall health of both the mother and the fetus. They can also be master deceivers and poor predictors of morbidity and mortality. They provide reassurance as well as signal impending doom and catastrophe. Pregnancy is characterized by several, normal physiologic changes within the body, and this is often reflected in the vital signs. These include blood pressure, heart rate, respiratory rate (RR), oxygen saturation, and fetal heart rate. It's important to understand what is considered to be "normal" vital signs in pregnancy since it's quite easy to do more harm than good.

## **BLOOD PRESSURE**

Blood pressure is the product of cardiac output multiplied by systemic vascular resistance (SVR). Rising levels of progesterone during pregnancy cause a decrease in SVR, which lowers blood pressure. This is seen during the first 16 to 18 weeks of gestation. At ~36 weeks, blood pressure slowly increases back toward prepregnancy levels.

As the uterus enlarges with the progression of pregnancy, it can physically compress the inferior vena cava (IVC) and lower portion of the aorta when in the supine position. This condition, known as aortocaval compression syndrome, can cause a decrease in blood pressure as large as 30 mm Hg and increases the risk of uteroplacental hypoperfusion.<sup>1</sup> Don't be

fooled into thinking this hypotension is due to shock from another cause. It can be easily fixed by repositioning the patient to the left lateral decubitus position, thus displacing the uterus off the IVC. If it's necessary to keep on a backboard, blankets can be placed underneath the right side of board in order to achieve a 15 degree angle of tilt. These patients should avoid lying completely supine for extended periods of time.

Pre-eclampsia is an important disorder of pregnancy characterized by elevated blood pressure and end organ damage, which can be in the form of Thrombocytopenia, Renal insufficiency, Intracranial disturbance, Proteinuria, LFT elevation, or pulmonary Edema (remember the mnemonic "TRIPLE"). Eclampsia is when patients with preeclampsia seize and can occur at any elevated blood pressure reading, regardless of whether it's mild, moderate, or severe.<sup>2</sup>

## HEART RATE

Heart rate increases ~10 to 20 bpm by the third trimester of pregnancy. This is mainly the result of increased blood volume and oxygen demand. As a result, changes in heart rate (and blood pressure) may not be seen until there is a blood loss of ~1.5 to 2 L. Tachycardia above the normal baseline heart rate for pregnant women may be a very late sign of hypovolemia from hemorrhage. To make things even more difficult, patients can in fact have a life-threatening diagnosis such as ruptured ectopic pregnancy in the presence of normal vital signs, thus giving it a poor negative predictive value when used as a diagnostic marker.<sup>3</sup>

Studies have looked into the use of "shock index" to predict the presence of ruptured ectopic pregnancy by detecting early hypovolemia. It is defined as the ratio of heart rate (in beats per minute) to systolic blood pressure (in mm Hg) with normal range being 0.5 to 0.7. In one particular study that looked at vital signs in women with known ruptured or unruptured ectopic pregnancies, those with shock index of 0.81 or higher were more likely to have ruptured.<sup>4</sup> The shock index utilizes the combined power of blood pressure and heart rate, since early acute blood loss of up to 450 mL is insufficient to produce changes in each vital sign alone.<sup>4</sup> With a much higher specificity (vs. sensitivity), this measurement can be utilized to "rule in" the likelihood of rupture.

## RESPIRATORY RATE/OXYGEN SATURATION

With normal progression of pregnancy, the diaphragm is pushed upward ~4



to 5 cm causing the lungs to shorten by the same amount, decreasing oxygen reserve. As the diaphragm elevates, the lower lobes of the lungs become more difficult to expand, leading to atelectasis. These changes predispose the patient to dyspnea and hypoxia at baseline.<sup>5</sup>

Pregnant women are more likely to desaturate quickly. Oxygen saturation may initially be normal, but take a turn for the worse with little to no warning. Airway management can be a challenge for even the most experienced physician, since upper airway edema caused by progesterone-mediated vasodilatation of mucosal vessels and reduced functional capacity place an increased oxygen demand.<sup>5</sup> Keep in mind that optimal fetal oxygenation is dependent on adequate maternal oxygen saturation.

RR during pregnancy remains essentially unchanged, but tidal volume (TV) increases. The increased TV causes overall minute ventilation to increase since it is the product of RR and TV. This leads to reduced PaCO<sub>2</sub> levels and places the patient in a chronic state of respiratory alkalosis with compensatory reduction in serum bicarbonate levels.<sup>5</sup> The good news is that more oxygen is delivered to the fetus (think of hemoglobin oxygen dissociation curve). The bad news is that lower bicarbonate levels at baseline places the pregnant patient at even higher risk of acidosis. Be very cautious when interpreting a “normal” PaCO<sub>2</sub> level in these patients. Normal in this case could represent impending respiratory collapse and should prompt the need for intubation.

## FETAL HEART TONES

Fetal heart rate should always be a part of the vital sign analysis of any pregnant patient presenting to the emergency department, regardless of their complaint.

### KEY POINTS

- Reposition pregnant patients to the left lateral decubitus position.
- Do not be reassured by normal vitals in a patient with suspected hemorrhage. By the time you notice hypotension or tachycardia, it may be too late.
- Don't forget about fetal heart rate as an important vital sign to check in pregnant patients.

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## **DON'T FEAR THE CORD!**

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Although recent reports indicate that US births approach 4 million annually, deliveries in the emergency department (ED) are relatively rare. Despite this, deliveries in the ED are precipitous, and emergency providers need to be familiar with the complications that may ensue. These births strike fear in the heart of many providers due in part to the rarity of their occurrence and the higher-risk population that presents to the ED for delivery, many of whom have received limited or no prenatal care. Familiarity with the management of obstetric emergencies including nuchal and prolapsed cords is crucial for emergency providers.

Nuchal cords are common, occurring in 15% to 34% of deliveries. Evidence suggests that most infants with a nuchal cord during delivery have favorable outcomes; however, potential catastrophic complications of nuchal cord include low APGAR scores, acidemia, and fetal demise. In the midst of the chaos of a vaginal delivery in the ED, it is crucial to remember to check for a nuchal cord after delivery of the infant's head. This is done by using an index finger to quickly but carefully feel around the infant's neck.

If a nuchal cord is palpated, it should be quickly but gently reduced by bringing it over the infant's head. In the case where a nuchal cord is too tight to reduce, the cord must be cut. This is done by placing two clamps several centimeters apart and carefully cutting the cord in between. Early clamping and cutting of the cord is not ideal as evidence shows that it can lead to neonatal hypovolemia, jaundice, and hyperbilirubinemia. However, in the emergent situation of a nuchal cord that cannot be reduced, it is a necessary procedure to allow completion of delivery.

Umbilical cord prolapse occurs rarely (up to 0.6% of births) but is life

threatening for the fetus secondary to compromise of blood flow from the umbilical vessels. Cord prolapse is defined as the presentation of the cord before the fetal presenting part (overt prolapse) or alongside the presenting part (occult prolapse). Cases of overt prolapse either will be visualized or will be palpated by the emergency provider during the vaginal exam. Occult prolapse is often not seen or felt on exam but should be suspected in cases of sustained fetal bradycardia. Regardless of type, the ultimate treatment is emergency cesarean section. Risk factors for umbilical cord prolapse that may be present in the ED patient include fetal malpresentation (transverse or breech), prematurity, multiparity, spontaneous rupture of membranes, and polyhydramnios. These increase the risk of prolapsed cord because the fetal head is not well engaged.

A sterile vaginal exam should be performed by the emergency provider expeditiously after the laboring patient's arrival. The purpose of the exam is to assess for cervical dilatation, rupture of membranes, and fetal presentation. At this time, it is vital to check for a prolapsed cord (which will usually feel like a soft, pulsatile mass). Even if there is no cord palpated initially, prolonged fetal bradycardia (fetal heart rate < 110) noted on the monitor should prompt the emergency provider to suspect cord prolapse and recheck the patient. It is possible that occult prolapse may be present even if no cord is palpated on exam although other causes of fetal bradycardia should also be considered such as vasa previa, placental abruption, and uterine rupture.

When a prolapsed cord is present, immediate steps should be taken to maximize uterine perfusion and thus perfusion to the fetus while waiting for an emergency cesarean section. The first step is for the examining provider to relieve pressure on the cord by manually lifting the presenting part off of the cord. This is done by applying enough pressure to the fetal head (or other presenting part) to lift it off of the cord using the examining fingers or entire hand. After the provider's hand is in place, the patient should be placed in Trendelenburg or knee-chest position, which will further relieve pressure on the cord. Next, a Foley catheter can be placed and 500 cc to 750 cc of normal saline infused into the bladder, which will help elevate the presenting fetal part off of the prolapsed cord. If fetal distress and bradycardia persist despite these temporizing measures, a tocolytic (terbutaline 0.25 mg) should be given. Fortunately, most infants do well after umbilical cord prolapse as long as perfusion is maximized by the measures listed above and cesarean section occurs within 30 minutes. In a small fraction of cord prolapse patients who are in the advanced stages of labor, it may be more expeditious to deliver the infant vaginally.

Deliveries in the ED are intimidating by their very nature and nuchal cord and prolapsed cord are two feared complications. However, emergency

providers need not fear the cord as long as they understand the necessary interventions.

## KEY POINTS

- Remember to check for nuchal cord after delivery of the head.
- If a nuchal cord is felt, attempt reduction, and if unsuccessful, clamp and cut the cord.
- The most important intervention for prolapsed cord is emergency cesarean section.
- Immediate steps to be taken for prolapsed cord include manual elevation of the presenting part, placing the patient in knee-chest position and Trendelenburg, and infusing normal saline into the bladder via Foley catheter.
- If fetal distress persists in the setting of cord prolapse, a tocolytic should be given.

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# TIMES A WASTIN': PERIMORTEM CESAREAN SECTION

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Greek god Apollo performed the first cesarean section on his wife, Coronis, while on the funeral pyre, birthing their son, Asclepius the demigod of medicine and healing. Rome's second king, Numa Pompilius, decreed that a child be excised from any woman who died in late pregnancy, to allow proper religious burial of both. Known as *lex regis de inferendo mortus*, the "perimortem cesarean section" first appears in 715 BC.

Historically, perimortem cesarean sections (PMCS) were performed for last minute fetal salvage when maternal mortality was inevitable. In total, medical literature describes just over 300 PMCS, with few in the last decade due to an increase in preventable maternal deaths and improved maternal health. Now, the most common cause of third trimester fetal death is maternal shock, carrying an 80% mortality.<sup>1,2</sup> In 1986, a published case series noted a surprising increase in the survival of moribund mothers who underwent timely PMCS with concurrent maternal cardiopulmonary resuscitation, leading to the current recommendations of the procedure.<sup>3</sup>

Key features of a successful PMCS include expeditious performance and adequate resuscitation of the pregnant woman. As such, an appreciation of the changes in cardiopulmonary physiology during pregnancy is essential. While cardiac output is increased overall, ~30% is diverted to perfuse the gravid uterus.<sup>4</sup> Additionally, around 20 weeks' gestation, the gravid uterus reduces venous return and distal aortic flow due to aortocaval compression. Oxygen consumption is higher in pregnancy, complicated by a reduced functional residual capacity and decreased oxygen-carrying capacity from dilutional anemia. Progesterone causes mucosa edema, while estrogen makes

tissues friable and hyperemic. Added to a laxity of connective tissues, a tougher intubation ensues. Finally, external cardiopulmonary compressions, which must be done higher on the sternum, only provide about a third of normal cardiac output. Therefore, early airway management and manual displacement of the uterus to the left lateral position optimize resuscitation efforts. Alternatively, a towel roll under the right hip or tilting the patient will work. Evacuation of the uterus—the cesarean section—optimizes maternal resuscitation in multiple ways and must be considered early in the course.

The two most important factors to consider in performing a PMCS are fetal viability and timing. Fetal survival with good neurologic outcomes is directly related to the time between maternal death and fetal delivery, with the best outcomes in those delivered within 5 minutes of maternal cardiac arrest. Current recommendations are to begin the PMCS by minute 4 of maternal pulselessness, completing fetal delivery within 1 minute of incision.<sup>3</sup> Of note, successful deliveries up to 30 minutes of cardiac arrest have been reported.<sup>5</sup> PMCS should still be considered if patients present late in their arrest. Secondly, PMCS should only be attempted on fetuses  $\geq 24$  weeks gestational age. Time is of the essence, thus dating by ultrasound is not recommended. A uterine fundus extending to the umbilicus is  $\sim 20$  weeks' gestation. Therefore, a quick and easy way to determine fetal viability of 24 weeks is the ability to palpate the fundus  $\sim 4$  fingerbreadths above the umbilicus.

Ongoing resuscitation efforts must occur concurrently with the PMCS for greatest success. Optimally, two teams of providers should be arranged, one each for the mother and neonate. Ideally, the assistance and expertise of an obstetrician and neonatologist should also be sought. If time permits, obtain vascular access above the uterus, prepare the abdomen, and decompress the bladder with a Foley catheter; however, do not delay for these!

Maximal exposure is *key* to rapidly access the fetus. Start with a vertical midline incision beginning from the xiphoid process extending to the pubic symphysis (cut around the umbilicus). Bluntly dissect the abdominal musculature to the peritoneum, which is incised vertically with scissors. To expose the uterus, retract any bowel or bladder from the visual field. Extract the uterus and vertically incise 5 cm through the lower uterine wall using scissors. With the other hand elevating the uterine wall away from the fetus, extend the incision upward until amniotic fluid is obtained or the uterine cavity is clearly entered. Cutting through an anteriorly placed placenta causes little harm. Find the head and disengage it from the pelvis if necessary. As in

a spontaneous vaginal delivery, suction the neonate, deliver the body, and clamp and cut the umbilical cord, retaining a segment for a cord gas. Premature infants are more likely to present in breech position, in which case, deliver the feet first. Separate and remove the placenta, ensuring the uterus is wiped out to prevent retention of placental products. Close the uterus with large, locking, absorbable sutures starting in the lower segment working cephalad and with layers as needed. The mother has the highest chance of return of spontaneous circulation at the moment of fetal delivery. Anticipate an increase in bleeding. Apply fundal massage and consider direct injection of oxytocin, carboprost, or methylergometrine to the myometrium prior to closing the remaining layers. Now, take a breath; you may have just saved a life, perhaps even two.

## KEY POINTS

- Don't be caught off guard.
- Identify the right situation for PMCS; obtain specialty assistance whenever possible.
- 24/4/4:  $\geq 24$  weeks' gestation, 4 fingerbreadths above the umbilicus, begin by 4 minutes.
- Continue CPR throughout the entire resuscitation, before and after delivery of the fetus.
- Know your policies; prepare and practice.

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# **CLOTTED LUNGS: NOT ALL SHORTNESS OF BREATH IN PREGNANCY IS FROM LAMAZE CLASS**

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Shortness of breath is not an uncommon complaint during pregnancy. Given the myriad number of physiologic changes associated with pregnancy, it would not seem unreasonable to attribute the dyspneic pregnant patient as merely experiencing a manifestation of these changes. However, the risk of pulmonary embolism (PE) in pregnancy is increased four- to fivefold when compared with nonpregnant women. In fact, PE is a leading cause of maternal mortality in developed countries. Knowing the presentation and proper testing for this condition is paramount in not only saving one life... but two!

## **PATHOPHYSIOLOGY**

Pregnancy is a complicated time for the human body. Pregnancy-related blood volume and vascular, hormonal, and mechanical changes have been associated with an increased risk of deep vein thrombosis. Venous stasis secondary to IVC compression by the uterus and decreased physical movement capabilities are the first part of the triad. Furthermore, a hypercoagulable state related to changes in multiple clotting factors (such as increased fibrinogen levels and decreased protein S) is produced. A prior history of DVT or thrombophilia increases this risk even more. The risk increases even further for patients undergoing a cesarean section delivery.

## PRESENTATION

The most common presenting signs of PE are tachypnea and tachycardia, with the “classic” symptoms being chest pain and dyspnea. Massive pulmonary embolism (such as the “saddle”-type PE) may be associated with syncope and low blood pressure, followed by PEA/asystole. A low index of suspicion is necessary in all cases. It is not uncommon for women to experience pregnancy-induced shortness of breath nor is syncope or near syncope uncommon in pregnancy. Heart rate is increased in the later stages of pregnancy (but not typically over 100). Coupling the constellation of signs and symptoms, while maintaining vigilance for the disease, will steer you clear of trouble and not miss this disease (fatal in 30% of untreated patients).

## DIAGNOSIS

Proper testing for diagnosing a PE in pregnancy includes modalities such as EKG, D-dimer testing, ultrasound, ventilation/perfusion (VQ) scanning, and helical CT pulmonary angiography (CTA). EKG-associated changes in PE include tachycardia (most common) and signs of right heart strain (the oft taught “S<sub>I</sub>Q<sub>III</sub>T<sub>III</sub>” findings of an S wave in lead I and a significant Q wave in lead III with an inverted T-wave in lead III, present in 20% of PEs, as well as T-wave inversions in the right precordial leads and incomplete right bundle branch block patterns). D-dimer testing may be performed, but varies in pregnancy, and is often positive. Yet, negative D-dimer assays are thought to maintain a reasonable negative predictive value at least for those low probability cases. Beginning with an ultrasound of the lower extremity is recommended first, particularly in patients presenting with symptoms of lower extremity DVT. Patients with a diagnosed DVT on ultrasound and associated symptoms of PE are assumed to have thromboembolic disease. Unfortunately, compression ultrasonography may miss pelvic deep venous thrombosis. A chest x-ray should be performed prior to CTA/VQ scanning. Patients with a normal chest x-ray are more likely to yield a diagnostic (less ambiguous) VQ study, and an alternative diagnosis (such as pneumonia) may be found. It is generally accepted that both VQ scanning and CTA represent low radiation risk to the fetus, and both are acceptable to rule out this potentially lethal disease entity. Given a normal CXR and no history of pulmonary disease, VQ scanning is preferred; otherwise, CTA of the chest is preferred. Patients with a nondiagnostic VQ scan with high index of suspicion for PE should have further testing in the form of CT pulmonary angiogram. Note ABG testing for A-a gradient abnormalities is not sensitive

in pregnancy and thus has limited usefulness.

Recent advances using magnetic resonance imaging for the diagnosis of PE has been promising. Although an ideal technique due to its lack of ionizing radiation, it lacks accuracy. This may change in the next few years as techniques advance to improve acquisition time, resolution, and motion artifacts. Unfortunately, gadolinium-based contrast agents have not been proved to be safe in pregnant patients and unenhanced MR imaging techniques are at present inaccurate for detecting clot in subsegmental pulmonary branches.

## TREATMENT

Warfarin is contraindicated in pregnancy. Treatment consists of low molecular weight heparin (LMWH) or unfractionated heparin based upon the patient's pre-/early pregnancy weight. Prior to initiation of treatment, blood should be taken for liver function testing, renal function, and baseline PT/PTT/INR. Thrombophilia screening in pregnancy may be inaccurate and is not routine. In massive PE, unfractionated heparin is preferred initially, with other and more extreme measures used on an individual basis and using a multidisciplinary team. With signs of cardiovascular collapse unresponsive to noninvasive management, thrombolysis should be considered via mechanical or using thrombolytic agents. IVC filters have a role in the management of recurrent DVT/PE despite anticoagulation or in patients that cannot use heparin products.

### KEY POINTS

- Pulmonary embolism risk is four- to fivefold higher in pregnancy.
- Presentation is similar to that of nonpregnant patients but with the confusion caused by crossover of benign similar symptoms in normal pregnancy.
- Diagnosis should start with bilateral lower extremity compression ultrasonography, EKG, and a chest x-ray.
- The most common EKG-associated abnormality is sinus tachycardia.
- If the patient has a normal chest x-ray and no history of prior pulmonary disease, and a nondiagnostic ultrasound, proceed with VQ scanning.
- Patients with an abnormal chest x-ray or a prior history of pulmonary disease should have a CT pulmonary angiogram of the chest instead of VQ scanning.

- Treatment for PE is with unfractionated heparin or LMWH. Warfarin is contraindicated.

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## **POSTPARTUM COMPLICATIONS**

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The postpartum female presenting to the emergency department can be a diagnostic challenge for the emergency provider. Beyond the common diseases and illnesses of this age group, postpartum physiology provides an additional layer of complexity for these women.

### **POSTPARTUM HEMORRHAGE**

Postpartum hemorrhage is often described in regard to timing: primary hemorrhage, often caused by uterine atony, within the first 24 hours following delivery, while secondary occurs between 24 hours and 12 weeks postpartum. Occasionally, emergency providers encounter patients who delivered within 24 hours prior to presentation. Initial evaluation should include a bimanual pelvic examination to assess for a “boggy” uterus. If found, consider compression of the uterus to expel blood and clots and help decrease bleeding. Examine for lacerations and hematomas as possible etiologies of bleeding. If more than 8 weeks postpartum, consider infection, retained products of conception (POC), bleeding diathesis, or choriocarcinoma as an etiology. Regardless, initial treatment begins with administration of uterotonic medications, such as oxytocin. Packing of the uterus can be considered as a temporizing measure, often in preparation for an exploratory laparotomy.

### **POSTPARTUM PREECLAMPSIA/ECLAMPSIA**

Although uncommon, postpartum onset of preeclampsia/eclampsia can be seen up to 6 weeks following delivery. Presentation can be atypical but often presents with new-onset hypertension and headache or blurry vision. Magnesium sulfate should be given to those at risk of developing seizures, in

addition to initiation of antihypertensive therapy.

## **POSTPARTUM FEVER AND INFECTION**

Fever is temperature of 38.0°C or higher. Common etiologies in the postpartum period include urinary tract infections (UTIs), wound infections, and mastitis. Postpartum women are at increased risk for UTIs secondary to several risk factors including catheterization, epidural anesthesia, and vaginal procedures. More frequently following cesarean births than vaginal delivery, endometritis usually develops within 5 days of delivery and presents with fever, uterine tenderness, foul lochia, and leukocytosis. Mastitis is often associated with breastfeeding and presents with fever, breast tenderness, warmth to touch, swelling, and skin redness.

## **VULVAR EDEMA**

Vulvar edema is frequently seen following vaginal delivery. Typical management includes ice pack and other comfort measures. Emergency providers need a high index of suspicion for patients presenting with increasing edema, induration, perineal pain, significant leukocytosis with left shift, and high fever; a more significant etiology of vulvar edema, such as infection including necrotizing fasciitis, can be present.

## **MENTAL HEALTH ISSUES**

Postpartum blues occurs in 15% to 85% of women within 10 days of delivery. Common symptoms include mood swings, irritability, tearfulness, fatigue, and confusion. Typically, this is a transient postpartum problem and does not require intervention; however, it is important to recognize as it is a risk factor for development of postpartum depression.

Postpartum depression is major depressive disorder starting within 1 month of child birth. A high index of suspicion is often needed to make this diagnosis as most new mothers feel guilt regarding the depressive symptoms and hide them from care providers. There are several risk factors for postpartum depression including history of depression or postpartum depression with previous pregnancies, family members with depression or mood disorders, increased personal life stressors such as job loss or pregnancy complications, a baby with health problems or special needs, and difficulty breastfeeding.

Postpartum psychosis is a psychiatric emergency frequently requiring inpatient hospitalization. It occurs in 1 out of 500 mothers, with rapid onset

of symptoms 2 to 4 weeks following delivery. Infanticide is one of most serious risks associated with this disease. Command hallucinations and the stressors of new infant care can increase the risk of infanticide after delivery in a mother with psychosis. Risk factors include history of postpartum psychosis, previous hospitalization for a manic or psychotic episode, recent discontinuation of mood stabilizers, as well as the risk factors associated with postpartum depression.

## **PERIPARTUM CARDIOMYOPATHY**

Development of decreased left ventricular ejection fraction in the last month of pregnancy or within 5 months of delivery is considered peripartum cardiomyopathy. Although rare, it can be lethal with progression to cardiac failure and sudden cardiac death.

Patients initially present with symptoms of mild heart failure, such as dyspnea and lower extremity edema, which is common in uncomplicated pregnancies and can be misdiagnosed by health care providers. Women can also have arrhythmias; embolic events due to the dilated, dysfunctional left ventricle; and acute myocardial infarction due to decreased perfusion to the coronary arteries. In the emergency department, treatment may include use of intravenous vasodilators and inotropic medications but may progress to needing more advanced circulatory support.

## **POSTPARTUM NEUROPATHY**

Postpartum neuropathy complicates 1% of deliveries. Typically resulting from compression, stretch, transection, or vascular injury, the most commonly injured nerve is the lateral femoral cutaneous nerve, but this can also be seen with the femoral nerve, peroneal nerve, lumbosacral plexus, sciatic nerve, and obturator nerve. Patients typically present with unilateral pain, weakness, and/or sensory abnormalities in the lower extremities, depending on affected nerve. Emergency providers should consider an alternative diagnosis if the patient has significant back pain, unexplained fever, or worsening neurologic symptoms. Anti-inflammatories are first-line treatment. Most women's symptoms resolve over days to weeks, with median time to resolution of 8 weeks.

### **KEY POINTS**

- Postpartum hemorrhage can be seen up to 12 weeks postpartum. Treat aggressively with fluid resuscitation, blood products, and uterotonic medications.
- Preeclampsia/eclampsia can develop in the postpartum period.
- Peripartum cardiomyopathy frequently presents with symptoms typical of pregnancy and requires a high index of suspicion to diagnosis.
- Mental health issues in postpartum women range from typical postpartum blues to postpartum psychosis with risks for both maternal and newborn health.
- Anti-inflammatories are first-line treatment for postpartum neuropathy, but consider a more sinister etiology of symptoms if associated with a fever, back pain, or worsening neurologic deficits.

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## THERE IS NO SINGLE TEST TO RULE OUT PID: JUST TREAT IT!

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### INTRODUCTION AND PATHOPHYSIOLOGY

The term “pelvic inflammatory disease” (PID) encompasses any combination of inflammatory disorders of the upper female reproductive tract, including endometritis, salpingitis, myometritis, parametritis, oophoritis, tuboovarian abscess, and pelvic peritonitis. It’s a tricky disease because while there is no historical, physical, or laboratory finding that is both sensitive and specific for the diagnosis, the long-term complications of mild or even asymptomatic PID can have devastating consequences on female reproductive health. It is **the leading preventable cause of infertility** and is linked to a large proportion of ectopic pregnancies. Missing the diagnosis and not treating is a high-risk decision; no one wants to be the reason a patient can’t have babies!

When you encounter a reproductive-age woman with any spectrum of lower abdominal pain in the emergency department, you must:

- 1) Have a high index of suspicion for PID
- 2) Treat suspected PID aggressively with antibiotics

### MAKING THE DIAGNOSIS

PID is a clinical diagnosis. Many episodes of PID go unrecognized because patients are asymptomatic or have nonspecific signs and symptoms. Patients may complain of lower abdominal pain, dyspareunia, malodorous or mucopurulent vaginal discharge, dysuria, abnormal uterine bleeding, or fever

and chills. However, when patients are symptomatic, your ED diagnosis of PID has a positive predictive value of up to 90% even compared with laparoscopy. That PPV increases if your patient falls within higher-risk groups: sexually active teenagers, women who have multiple male sex partners, women who attend STD clinics, women who have had PID before, women in lower socioeconomic groups, and women who live in areas with high rates of gonorrhea and chlamydia.<sup>1</sup>

There are physical exam findings that indicate PID, and they are *not* limited to cervical motion tenderness. Any of these findings—cervical motion tenderness, uterine tenderness, or adnexal tenderness—may be enough to initiate empiric treatment for PID. Remember that other pathologies, including ectopic pregnancy, appendicitis, diverticulitis, urinary tract infections, and ovarian cysts, can all cause tenderness to palpation on a bimanual exam. Additional findings of fever, mucopurulent vaginal discharge, a friable cervix, a saline prep (wet mount) of vaginal fluid showing many WBCs, and documented gonorrhea/chlamydia infection all increase the specificity of your clinical diagnosis of PID.

Most patients in whom you suspect PID should undergo a transvaginal ultrasound to look for tuboovarian abscess, free fluid, tubal hyperemia, ectopic pregnancy, and ovarian torsion. All patients diagnosed with PID should be tested for pregnancy, gonorrhea, and chlamydia. HIV testing in nonhospitalized patients should be done in the ED or outpatient setting, depending on site-specific resources available for follow-up of results. “But hey,” you’re thinking. “My patient was tested negative for STDs the other day.” Actually, fewer than 50% of women with PID have a positive test for *N. gonorrhoeae* or *C. trachomatis*, and a negative endocervical swab can’t rule out upper reproductive tract infection by those organisms. As if that wasn’t enough, nonsexually transmitted vaginal flora and other lower urogenital tract bacteria also cause PID.

Bottom line: Just treat it.

## TREATMENT

Preventing complications of PID depends on early antibiotic treatment. Appropriate treatment consists of adequate coverage for gonorrhea, chlamydia, and anaerobes for both the patient and sexual partners. Oral antibiotics are equivalent to IV for mild to moderate disease,<sup>2</sup> and treatment generally takes 14 days. Refer to the CDC’s STD treatment guidelines (below) for up-to-date information on recommended regimens. There is also a handy app (CDC STD TX Guide) available free for download to your

mobile device.

In addition to return precautions, discharge instructions should include abstinence from sex until after patients and their partners complete treatment. Follow-up with a gynecologist should happen after 3 days, and testing for gonorrhea and chlamydia should take place again 3 months after treatment or if the patient presents similarly within 12 months.<sup>1</sup>

Patients that require admission:

- Can't exclude surgical emergency
- Tuboovarian abscess
- Pregnancy (high risk for maternal morbidity and preterm labor)
- Sepsis or ill appearance
- Failed outpatient therapy (i.e., no symptomatic improvement after 72 hours of outpatient treatment)

## SPECIAL CONSIDERATIONS

Patients with HIV and PID are more likely to develop tuboovarian abscesses, though they respond to antibiotics similarly to non-HIV patients. Patients with intrauterine devices are at highest risk of developing PID within the first 3 weeks after insertion. If a patient with an IUD presents with PID, the IUD does not need to be removed unless the patient has not improved after 3 days of treatment.<sup>3</sup>

Fitz-Hugh-Curtis syndrome (FHC) is perihepatic inflammation of the liver capsule that causes sudden, severe, right upper quadrant pain, tenderness to palpation, fevers, chills, and malaise. Bimanual exam, RUQ ultrasound, and CT scan of the chest or abdomen may be negative. Many times, the only clue to diagnosing FHC is the knowledge or suspicion that the patient has gonorrhea or chlamydia with no other demonstrable cause for RUQ pain. FHC is treated with the same antibiotic regimen used for PID. If pain persists, surgery may be required to remove liver capsule adhesions.

## KEY POINTS

- A negative STD screen does not rule out PID.
- Initiate empiric treatment for any young, sexually active woman with an exam suggestive of PID—and treat partners, too.
- All women diagnosed with PID should be tested for pregnancy, HIV, gonorrhea, and chlamydia.

- PID + pregnancy = hospital admission.
- PID with IUD = the IUD can stay in, but patient should have gynecologic re-evaluation within 72 hours to assess for improvement on treatment.

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# **DON'T DISMISS THE YOUNG, FEMALE PATIENT WITH SHORTNESS OF BREATH WITHOUT CONSIDERING PERIPARTUM CARDIOMYOPATHY**

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**KATHI GLAUNER, MD, PhD**

Your patient is a 33-year-old woman who presents with shortness of breath and cough. She denies medical history, takes no medications, and doesn't smoke. She is tachypneic with a productive cough. You evaluate her and note crackles in her lung bases. You diagnose pneumonia and send her home with antibiotics because you believe she is low risk for poor outcome.

Patient number two is a 28-year-old woman with shortness of breath and bilateral leg swelling. She is 38 weeks pregnant. This is her first pregnancy. She is otherwise healthy. You explain to her the normal expected changes in late pregnancy, including increased blood volume, lower extremity edema, and shortness of breath due to increased pressure from the distended abdomen. You send her home with congratulations and return precautions.

Both patients later return with signs of decompensated heart failure. They are rapidly intubated and sent to the ICU on nitroglycerin drips and dobutamine infusions. They are diagnosed with peripartum cardiomyopathy (PPCM) based on their echocardiogram findings. Where did you go wrong? How can you avoid this mistake in the future?

Your first task is to recognize the possibility of PPCM in young and

healthy patients. PPCM is a potentially deadly type of heart failure found in women of child-bearing age. By definition, PPCM occurs in late pregnancy or the early postpartum period. The condition is relatively rare, occurring in only ~1 in 4,000 pregnancies in the United States. Untreated, it has a 5% to 30% mortality rate.<sup>1</sup> Because PPCM is uncommon, it can be easily missed. However, its severity dictates that it always be included in the differential for women of child-bearing age with shortness of breath. Always ask about recent pregnancy to avoid missing this illness in the postpartum woman.

PPCM is of unknown etiology, although recent studies have implicated abnormal prolactin levels as a potential cause. It has also been suggested that myocarditis plays a role in development of PPCM. PPCM is more frequent in women >30 years old, multiparity, women with a history of hypertension, and those with prolonged use of tocolytics. There is high risk of recurrence in women who become pregnant again after having PPCM. It is important to remember, however, that women without these risk factors can also develop PPCM.<sup>2</sup>

Women with PPCM present with signs and symptoms of heart failure. They may complain of shortness of breath, leg swelling, fatigue, or cough. Other symptoms may include paroxysmal nocturnal dyspnea (PND) or hemoptysis. The diagnosis is frequently missed because the symptoms overlap with some of the typical late pregnancy or postpartum symptoms.<sup>2,3</sup>

On physical exam, patients may have murmurs, jugular venous distention or a third heart sound. They could have crackles on pulmonary auscultation as well as lower extremity edema. Again, some of these signs overlap with the normal changes of pregnancy and are easily discounted by a less cautious physician. Fairly simple testing can help guide you in your diagnosis.

The diagnosis of PPCM can only be made by excluding other causes of these symptoms. Investigation should include a chest x-ray, electrocardiogram (ECG), echocardiogram, troponin, beta natriuretic peptide (BNP), thyroid-stimulating hormone (TSH), complete blood count (CBC), and comprehensive metabolic panel (CMP). This allows evaluation for myocardial infarction, electrolyte and metabolic disorders, pulmonary embolus (PE), and other concerning syndromes in pregnancy such as HELLP and pre-eclampsia/eclampsia. Elevation of BNP and evidence on echocardiogram of depressed left ventricular (LV) function help make a diagnosis of heart failure.

The chest x-ray may show pulmonary edema or cardiomegaly but otherwise be unremarkable. ECG likewise may have nonspecific findings including sinus tachycardia or left ventricular hypertrophy. While BNP is

sometimes mildly elevated in pregnancy, it may be greatly elevated in PPCM.

To say with more certainty that this is PPCM, the patient must fulfill three criteria: (1) she must be in late pregnancy or the early postpartum period, (2) must have new-onset heart failure without another cause, and (3) have LV dysfunction, usually with LVEF < 45%.<sup>3</sup>

Management of women with PPCM is similar to that of any person with heart failure, with the goal of stabilizing heart failure. However, fetotoxic medications should be avoided. Immediate treatment in the ED includes use of inotropes and preload/afterload reducers such as dobutamine or milrinone and nitroglycerin.<sup>4</sup> Use caution with loop diuretics and ACE Inhibitors (ACEIs) in pregnancy as loop diuretics can decrease blood flow to the fetus and ACEIs are teratogenic. These are standard therapy, however, in postpartum women. Decompensated patients may require intubation and ventilation. Arterial lines to monitor blood pressure and administration of nitroglycerin and dobutamine to regulate preload and inotropy will be important treatments. Heparin to prevent embolic events should be initiated. Both compensated and decompensated heart failure patients should be admitted for treatment and stabilization of new-onset cardiomyopathy.<sup>5</sup>

In summary, recognition will be the most important step in diagnosis of PPCM. Evaluate even young and presumably healthy patients carefully with BNP, CXR, EKG, and echocardiogram. Once diagnosed, treat their heart failure aggressively and admit to the hospital for further treatment.

## KEY POINTS

- Recognition is the most important step in diagnosis of PPCM.
- PPCM should be considered in all late term pregnancy and postpartum patients.
- The diagnosis of PPCM can only be made by excluding other causes of these symptoms.
- Management of PPCM is similar to that of any person with heart failure, with the goal of stabilizing heart failure.

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# SECTION XV

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## PSYCH

## DELIRIUM

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CASEY CARR, MD

Delirium is a capricious and varied disorder—with equally varied etiologies. Delirium is very common, occurring in up to 30% of hospitalized patients, and is most common in the elderly. This predilection for the hospitalized elderly, who are already at risk for acute illness, creates the potential for diagnostic confusion and increasing morbidity and mortality risk. Delirium is traditionally defined as an acute change in cognitive functioning and sensorium, usually secondary to an underlying medical illness. The key features of delirium include disturbances in consciousness, attention, cognition, and perception that occur over a truncated period of time. This change in mental status tends to fluctuate throughout its course. Please refer to the APA's DSM V criteria on delirium. The pathophysiology behind the mechanism of medical illness that causes an acute change in cognition is poorly understood. Theories include neurotransmitter abnormalities in response to systemic insult and transient occult brain injury. Risk factors for developing dementia include hospitalized elderly, multiple medical conditions, terminally ill, children, multiple medications, and sensory deprivation.

## DIAGNOSIS

There are myriad causes of delirium—and an equally large number of memory aids on the same topic—however, there are certain etiologies that an emergency physician must be facile at identifying. However, initial assessment should be focused on the actual presence of delirium and if a patient's change in mental status is secondary to an alternative cause. Psychiatric illness and dementia are possible causes of changes in mental status that may mimic delirium—focus should be on elucidating the defining details between these disorders. Timing and onset is a crucial indicator of

delirium; acute onset and a fluctuating mental status are common with delirium but uncommon in other conditions. Common tools to assess a patient's mental status are both the Mini-Mental State Examination and the Confusion Assessment method. Etiologies of delirium can be broadly categorized as central nervous causes, metabolic disorders, cardiopulmonary disorders, and systemic illness; these categories are more completely elucidated in [Table 212.1](#). A well-established mnemonic for the causes of delirium is I WATCH DEATH, standing for **I**nfection, **W**ithdrawal, **A**cute metabolic changes, **T**rauma, **C**NS disease, **H**ypoxia, **D**eficiencies, **E**nvironmental, **A**cute vascular changes, **T**oxins, **H**heavy metals. Diagnostic interventions should aggressively search out these causes, since treatment is based on treating the underlying cause. A focused history and physical with basic laboratory studies—including urine analysis—is an appropriate initial step in management.

**TABLE 212.1 COMMON ETIOLOGIES OF DELIRIUM**

|                      |   |
|----------------------|---|
| CNS disorders        | Vascular abnormalities, compressive lesions, postictal state, intracranial hemorrhage         |
| Metabolic disorders  | Electrolyte abnormalities, uremia, hepatic encephalopathy, hypoxia, hypoglycemia, Wernicke's  |
| Infection            | Urinary tract infection, pneumonia, meningitis, sepsis  |
| Environmental causes | Sensory deprivation, postoperative state, polypharmacy, substance abuse, substance withdrawal |
| Systemic illness     | Myocardial infarction, pulmonary embolism, toxic ingestions, heavy metal poisoning            |

## TREATMENT

Treatment of delirium should be focused on treating emergent etiologies of delirium. Early goal-directed therapy should be the first goal when managing a patient with delirium. However, a commonly encountered challenge is the decision as to when to intervene when a patient becomes increasingly combative and agitated. The first step in managing the agitation of delirium is environmental interventions. Reduce the disruption to the patient's wake-sleep cycle—sensory deprivation with dark, quiet surroundings during the night and regular modest amount of stimulation during the day. Physical impairments of patients worsen delirium, so utilizing the patient's own glasses and hearing aids is a must. Any step that increases the patient's familiarity with the surroundings will reduce the behavioral burden of delirium, especially in the elderly. Encourage regularity of tasks that need to

perform with the patient. Chemical and physical restraints should be kept to a minimum.

## KEY POINTS

- Know the risk factors for patients at risk for delirium—age, poor cognitive function, poor functional status, sensory impairment, coexisting medical illness, and polypharmacy.
- Never assume your patient’s agitation is due to dementia or psychiatric disease—always keep delirium on your differential.
- Know the natural course of delirium—acute onset, fluctuating course, inattention, disorganized thinking, perceptual disturbances, emotional disturbances, and altered sleep-wake cycle.
- Know the common etiologies of delirium and those that require immediate intervention in the emergency department.
- Implement environmental interventions for behavioral control in the emergency department—avoid using chemical and physical restraints.

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## **RESTRAINT WITH RESTRAINTS: PATIENT RESTRAINT**

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**GRANT NELSON, MD AND ROBERT B. DUNNE, MD,  
FACEP**

The decision to restrain a patient in the emergency department (ED) is often challenging. The main indication for restraint is protection, whether for the patient or the staff. An altered, thrashing patient can pose a very real threat to themselves and the staff in an ED. Agitation is a form of delirium, and the cause must be actively sought and addressed. Central nervous system lesions, post-ictal state, hypoxia, sepsis, medications, and substance related disorders are just a few of the causes of agitation, and prompt management of these conditions can facilitate diagnosis and treatment. The initial treatment for agitation is usually verbal deescalation and/or seclusion in a quiet room. Many times this is all that is needed to reestablish normal communication. When verbal counseling and isolation are not enough to deescalate a dangerous situation, a decision to restrain the patient should be made before further harm can occur. In these situations, it is imperative to document that (1) an emergency existed, (2) there was an inability to get consent, and (3) the treatment was for the patient's benefit.

Chemical sedation is often preferable to physical restraint as it avoids many of the potential consequences of physical restraints such as injury, rhabdomyolysis, hypoxia, and hyperthermia. In practice, physical restraint of some kind is often necessary as a bridge to chemical restraint. The end goal is a relaxed patient who can systematically have the physical restraints removed and participate in their own care. Any time physical restraints are contemplated, and chemical sedation should be considered. Physical restraint should rarely be used in isolation.

A team effort is required to restrain a patient, and the more hands available, the quicker and safer the process will be both for the staff and the patient. Often a “show of force” in the form of additional personnel is all that is required for a patient to quiet down and become more compliant. Ideally there should be one person for each extremity and one person designated to rapidly apply hard restraints. Though soft restraints work well for the mildly disoriented patient trying to pull at a line or tube, they are not appropriate for agitated patients. Hard restraints should be used on all four extremities to begin with and should be tightened to allow minimal movement. This will ensure the restraints will not fail, and the patient’s range of movement will be predictable. The hospital staff can then further assess and treat the patient more safely. Patients in restraints should be placed on the monitor and require constant observation.

Once the patient is in hard restraints, the goal should be to add chemical sedation as soon as possible to avoid adverse events and get the patient reengaged to their own treatment. The ideal chemical restraint drug is absorbable via intramuscular (IM) injection as this prevents the need to obtain intravenous (IV) access in an agitated patient. A coached patient can often cooperate with oral medication administration once they are redirected. Decisions regarding medication should be based on differential diagnosis of underlying cause. A patient whose initial complaint is that they are out of their antipsychotic medication and then becomes agitated may benefit from different medications than a patient with undifferentiated delirium.

Benzodiazepines are a mainstay of treatment, and a good first choice, thanks to their relative predictability and side effect profile. Lorazepam 1 to 4 mg IM or IV and diazepam 5 to 10 mg IM or IV are the two most traditionally used benzodiazepines. Midazolam IM is the most common prehospital medication used for agitation and has been shown to be the most predictable and rapid acting medication with 5 mg IV or 10 mg IM reliably calming most patients. Benzodiazepines should be titrated to effect, and caution should be used in the elderly and the alcohol intoxicated to prevent oversedation. In some patients, benzodiazepines can cause a disinhibition effect that could exacerbate or even cause an acute confusional state.

Typical antipsychotics such as haloperidol (2.5 to 5 mg IM) are commonly used as an initial strategy, often in combination with a benzodiazepine, and have been shown to be safe in many studies. It is important to remember that all antipsychotics have the potential to cause QT prolongation. There is potential in cases of unknown ingestion to exacerbate already present cardiac dysfunction, and it is prudent to avoid antipsychotics in those cases to prevent decompensation.

The atypical antipsychotics have proven to be useful and benefit from causing fewer extrapyramidal symptoms (EPS) than the typical antipsychotics. The most common atypical antipsychotics used in the emergency department are olanzapine (Zyprexa) and risperidone (Risperdal). Though risperidone is only available in oral formulation, it has been shown to be as effective as IM haloperidol and is a good choice in a patient who is agreeable to oral medications. Olanzapine has been looked at more recently as a potential alternative to haloperidol and has shown similar efficacy in agitation with less risk of EPS. There are numerous small studies assessing ketamine use in agitated patients, particularly in the prehospital setting. Ketamine IM is rapidly effective and has been shown to be safe, but more studies need to be done to assess its use in the ED.

There are countless cases where the initial impression of the cause of agitation was misguided and patients subsequently decompensated. Careful full physical examination is essential as is seeking additional history when possible. Agitated patients must be continually reevaluated. Always be aware of the safety of the patients and the staff. Whether from agitation from intoxication or medication, hypoxia, or shock, it's important to recognize when someone has the potential to become dangerous to either themselves or others.

## KEY POINTS

- Don't hesitate to use restraint to protect an altered, agitated patient from harming themselves or harming the staff if attempts at verbal deescalation and seclusion have failed.
- Initial therapy with IM midazolam may be the most reliable to sedate an extremely agitated patient to facilitate initial evaluation.
- Haloperidol and lorazepam together or alone are fast acting and safely reliable for longer sedation.
- Use lower starting doses in pediatric, intoxicated, and elderly patients to prevent oversedation.
- All restrained patients should be on a cardiac monitor and be frequently reevaluated with appropriate documentation.

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## MENTAL STATUS CONCERN? CONSIDER PSYCHOSIS

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**SARIKA WALIA, MBBS AND SHABANA WALIA, MD**

Psychosis is best understood as a syndrome. The term is used to describe a nonspecific group of signs and symptoms that impair one's ability to understand reality. It combines medical and psychiatric causes for a patient's difficulty in understanding his or her surroundings. Psychosis is not an illness in itself but occurs secondary to a broad array of psychiatric and medical illnesses. When a physician believes a patient may be psychotic, they must search for an underlying cause in order to discover the etiology of the patient's symptoms.

Psychosis can present with delirium, agitation, delusions, paranoia, auditory or visual hallucinations, confusion, or an altered mental state. There are two classifications for psychosis: functional and organic. While functional psychosis has not been fully understood, it is commonly known as psychosis without a clear medical cause. It is often broadly subdivided into schizophrenia, bipolar psychosis, and mood disorders with psychosis. The diagnosis of functional psychosis tends to be a diagnosis of exclusion, especially on first-time presentation. After medical causes have been ruled out, trials of therapeutic treatment can be made and response to the treatment confirms the diagnoses of these psychiatric illnesses.

Organic psychosis refers to abnormal mentation caused by a medical or physical cause, rather than a psychiatric illness. It is sometimes referred to as "organic brain syndrome" and can further be divided into delirium versus dementia. Common causes of emergency department visits for organic psychosis include cerebrovascular disease, endocrine disorders such as thyrotoxicosis and thyroid storm, substance abuse, drug or alcohol withdrawal, hepatic or metabolic encephalopathy, trauma, polypharmacy,

and sepsis. One can imagine, if a patient is severely hypoxic or hypercarbic, they can present altered or even delirious and until full medical evaluation occurs, they could be falsely diagnosed with a psychiatric illness. Dementia is another common reason for organic psychosis which is defined as a progressive decline in memory or cognition such as Parkinson disease, Alzheimer disease, Lewy-body dementia, or vascular dementia.

Varying presentations of psychosis will often point to a certain illness. For example, a patient with delusions (fixed beliefs that are false) in conjunction with antisocial behavior and disorganized thinking would be a common presentation of schizophrenia. However, a patient with visual hallucinations of bugs crawling all over their skin who is tachycardiac and tremulous may be suffering from delirium tremens secondary to alcohol withdrawal. The emergency physician must take into account the history, physical exam, and complete clinical picture in order to correctly identify the cause of psychotic behavior.

The emergency physician must be able to identify acute psychosis, differentiate between organic and functional psychosis, regulate the patient's behavior, and treat the life-threatening causes of psychosis. The approach to the psychotic patient should be similar to that of all other patients in the emergency department. Below is a step-wise approach in evaluating a patient with psychosis.

- 1) ABC's: Ensure airway, breathing, and circulation are intact.
- 2) The patient should be approached in a nonthreatening manner in a quiet and calm setting. The need for chemical or physical restraints in order to obtain a primary and secondary survey must be carefully thought out.
- 3) History from patient and family/friends: Collateral information from police, family, close friends, and coworkers will aid in obtaining information about the patient's medical history and baseline mental state. It is often helpful to evaluate if the symptoms were acute or gradual in onset, if the patient has a history of psychiatric disease, and to obtain a full medication list.
- 4) Physical exam including a mental status exam: Always undress each patient to look for signs of trauma as a medical cause for change in mental status.
- 5) Laboratory and Imaging: For first-time psychosis, labs such as thyroid studies, ammonia, urinalysis, electrolytes, urine toxicology, and a possible noncontrast CT of the head should be ordered.
- 6) Observation and frequent monitoring of vital signs.

Following the system above, the emergency physician can determine whether a patient has an organic or functional disorder and thus initiate treatment rapidly if needed. A study conducted by Dubin et al. 1983 showed that there are four factors that favor diagnosis of organic psychosis versus functional psychosis: abnormal vital signs, new memory loss, patient age >40 with no psychiatric history, and disorientation with a clouded mental status. Acute psychosis is an emergency. Most organic psychosis will require admission until symptoms resolve or are treated appropriately, and functional psychosis will often require admission as these patients are usually deemed a harm to themselves, society, or they do not have a reliable and safe disposition.

## KEY POINTS

- Psychosis is a nonspecific group of signs and symptoms that cause a patient to lose touch with reality.
- Psychosis can be associated with every organ system and thus warrants medical workup, especially on first-time presentation.
- Psychosis in patients with prescribed medications can often be a result of polypharmacy or as a result of substance abuse or drug withdrawal.
- Organic psychosis is caused by physiologic illness that affects the brain, whereas functional psychosis is caused by a psychiatric illness, but physicians must remember that functional psychosis is a diagnosis of exclusion.
- The emergency medicine physician must differentiate between organic and functional psychosis with a proper initial workup and evaluation in order to treat the patient correctly.

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## ASK ABOUT SUICIDE RISK

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**TRENT R. MALCOLM, MD, MS AND DONALD W. ALVES,  
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### CASE

*A 24-year-old homeless man with a history of bipolar disorder, polysubstance abuse, and alcohol abuse presented to the ED with acute alcohol intoxication and suicidal ideation. The patient reported worsening depression since running out of his medications several weeks prior. He was withdrawn and reported hopelessness with his current situation. When directly questioned, he endorsed a plan to step in front of a moving bus. He had been hospitalized in the past for severe depression and reported at least one prior suicide attempt by intentional drug overdose. The patient was observed for 12 hours and then reassessed. After sobering, he recanted his suicidal ideation and requested discharge.*

Evaluation of the suicidal patient is an inherently high-risk situation. Fundamentally, we must distinguish the patient that is an *imminent risk* to self from one that can be safely discharged. This is no small task, especially with a patient whom you have no prior relationship, working in a busy department with limited psychiatric resources. Nearly every emergency practitioner can cite a horror story of a patient discharged from the emergency department only to commit suicide within a matter of hours. Short of hospitalizing every suicidal patient, there is no way to completely safeguard against this nightmare scenario. However, employing a rational,

standardized approach to the suicidal patient will help minimize risk and improve patient care.

Suicidal risk assessment is a highly subjective task that demands a thorough history. The challenge of gathering a detailed and accurate history is routinely compounded by a number of patient factors, including emotional lability, mistrust, and intoxication. Yet, several key elements of the history are essential for an informed risk assessment. All suicidal patients should be asked about past psychiatric history, which includes psychiatric hospitalizations (e.g., number, timing, duration, and reason for admission) and suicide attempts (e.g., number, timing, method, and health effects). Additionally, all patients should be asked about recent life stressors, feelings of depression or hopelessness, and social support networks. There is no evidence that asking direct questions about suicide will introduce or worsen suicidal thoughts. It is therefore essential to be direct about recent or ongoing suicidal ideation (“Have you thought about killing yourself?”), plans (“How would you do it?”), and intent (“Would you kill yourself if you left the hospital today?”). Finally, every patient should be asked about access to firearms or other lethal means to carry out the stated plan. Quite simply, if you don’t ask, you’ll never know the answer.

Alcohol intoxication in the suicidal patient frequently complicates an already complex situation. Alcohol abuse is a major lifetime risk factor for suicide, and acute alcohol intoxication increases the immediate probability of suicide in at-risk patients. In one study, one-third of completed suicides had elevated blood alcohol levels at autopsy. Yet, patients with suicidal ideation while intoxicated frequently recant these feelings once sober. Consequently, many practitioners insist on sobriety prior to initiating an evaluation. Some centers require the alcohol level fall below a specific cutoff (e.g., 0.08 or 0.1 g/dL) prior to assessment. However, such requirements may unnecessarily delay disposition. According to ACEP guidelines, the decision on when to commence a psychiatric assessment should be based on the patient’s cognitive function, not alcohol level. This is especially important in chronic alcoholics for whom delaying evaluation until the blood alcohol level falls below the legal driving limit is inviting onset of withdrawal symptoms. Regardless of when the assessment is initiated, a suicidal patient should never be discharged while intoxicated, even if the patient has recanted.

Risk factors for suicide are well documented. Severe depression, feelings of hopelessness, and prior suicide attempts are widely regarded as the strongest predictors of suicidal behavior. Some other key risk factors include age (adolescents and elderly), gender (females are more likely to attempt, but males more likely to complete), substance and alcohol abuse, organized plan, comorbid psychiatric illness, family history of suicide, impulsivity, recent

unemployment, recent incarceration, recent initiation of antidepressant medication, social isolation (e.g., never married, separated, or divorced), and chronic illness. The emergency provider should consider these risk factors in the overall risk assessment for every patient, every time. Several clinical tools, including the SADPERSONS scale and Suicide Intent Scale (SIS), are widely used to aid in suicide risk assessment. However, providers are cautioned against relying on tools as few are validated for use in the clinical environment. The SADPERSONS tool has low sensitivity and cannot predict future acts of self-harm better than chance alone. Unfortunately, there is no substitute for a detailed, individualized assessment.

Some providers enjoy the luxury of a psychiatric consultant, but the vast majority must conduct these critical assessments on their own. When working with a psychiatric consultant, it is essential to remember that they are just that—a consultant. Ultimately, the decision to admit or discharge (and the legal responsibility for patient outcomes) rests with the primary provider. Therefore, it is essential to have detailed conversations with consultants to understand the reasoning behind their recommendations. When opinions differ, remember to never discharge a patient you feel will be unsafe. Finally, it is critical to document all pertinent aspects of the history including changes in mood and suicidal ideation, evidence of intoxication or sobriety, discussions with consultants, and the thought process leading to an overall risk assessment. If it isn't on paper, it didn't happen.

## KEY POINTS

- Gather a detailed history; if you don't ask the question, you'll never know the answer.
- Never discharge an intoxicated patient with a chief complaint of suicidal ideation.
- Assess for key suicide risk factors in every patient, *every time*.
- Trust your psychiatric consultants, but don't substitute their judgment for your own.
- Document, document, document! If it isn't on paper, it didn't happen.

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# **STRANGE BEHAVIOR? PERSONALITY DISORDERS IN THE ED**

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**KERRI N. BOOKER, RDH, MMS, PA-C**

Emergency medicine providers see a large number of patients with psychiatric complaints. It is the responsibility of the provider to ensure that any acute medical conditions are addressed to determine the underlying etiology of the patient's symptoms. Many patients seen in the emergency department (ED) have personality disorders, which may be undiagnosed or unknown to the patient. Providers must understand the core features of such disorders to recognize affected patients and provide them with the best possible care.

Personality disorders are defined as ingrained and pervasive patterns of maladaptive behavior, leading to impaired functioning. The very fact that these behavioral patterns are inflexible and ingrained within the patient presents a very unique challenge for the treating clinician. The difficult task of treating specific personality disorders is most appropriately handled by psychiatrists and other mental health professionals after medical clearance has been issued for the patient. However, emergency medicine providers are often the first point of contact for these patients. The American Psychiatric Association has classified personality disorders into three clusters based on prominent characteristics which can be more easily remembered and applied by the front-line provider in treating the patient.

Cluster A ("odd") disorders include schizoid, paranoid, and schizotypal personalities. Schizoid personality types tend to be withdrawn, introverted, and avoid close relationships. Paranoid personality types tend to be overly

sensitive, suspicious, and defensive. Schizotypal personality types are also suspicious, often superstitious, and socially isolated. They may also display eccentric behaviors and speech. Patients with cluster A disorders, especially paranoid types, are difficult to interact with and treat because they may perceive aggression and hostility in others, even when it is not present. Schizotypal personality disorder differs from the other cluster A disorders in that patients tend to display more bizarre behaviors such as dissociation, magical thinking, or clairvoyance. Cluster A patients generally demonstrate an enduring pattern of distrust and suspicion of other people and their motives. They are often difficult to engage and uncomfortable in interpersonal situations. It is important to remain professional, yet empathetic toward their fears. To improve communications with these patients, provide clear explanations of their treatment plan and your role in it, and avoid overinvolvement in their social or personal issues. It is very important not to react emotionally to their behavior. For patients with paranoid personality disorder, do not attempt to challenge their paranoid ideas or become distracted by them. Rather, allow the patient an opportunity to describe their ideas and simply move on.

Cluster B (“dramatic”) disorders include borderline, histrionic, narcissistic, and antisocial personality disorders. Patients with borderline personality disorders tend to be the most severely affected as they tend to be in conflict with society. They may be impulsive and aggressive, lack self-control, and have unstable personal relationships. These patients also display a high incidence of suicide attempts and drug abuse. Histrionic types are often vain, emotionally labile, and display immature, seductive, and egocentric behavior. Narcissistic types are often preoccupied with power, demand attention, and display exhibitionist or grandiose behavior, while showing a lack of interest in others. Antisocial types tend to be selfish, callous, and often have difficulty learning from prior experiences. They can be impulsive and promiscuous, which frequently leads to legal problems. With cluster B disorders, it is important to communicate clearly while setting limits during the conversation. Avoid overtechnical explanations, and once again, do not react emotionally to outbursts or dramatic behavior. Acknowledge the patient’s concerns, and give simple, factual responses to questions. Because these types of patients tend to have more behavioral relationship issues, it is very important to document your interactions carefully. A carefully documented medical record may be helpful in understanding and managing the patient at a later time.

Cluster C (“anxious”) disorders include obsessive compulsive, dependent, and avoidant personality disorders. Patients with cluster C disorders are considered the most highly functioning compared to their

cluster A and B counterparts. Patients with obsessive compulsive personality disorder (OCPD) are often perfectionists, egocentric yet indecisive, and possess rigid thought patterns and a need for control. Dependent types display a lack of confidence, poor self-esteem, are often overaccepting, passive, and unable to make decisions. Avoidant types fear rejection, have low self-esteem, and hyperreact to failure or rejection. Once again, validate their concerns while providing reassurance and setting limits in your involvement. This is especially pertinent in the case of dependent personalities; remember, you cannot be everything to everyone. For OCPD patients, perform a very thorough history and exam, provide them with comprehensive explanations of their treatment, and encourage their participation. Avoidant types may require extra encouragement to report their concerns, symptoms, and reasons for their presentation to the ED.

Patients with personality disorders often meet the criteria for more than one disorder and frequently have multiple comorbid conditions including drug and alcohol abuse, anxiety, and depression. They may have fixed perceptions of the world as unsafe and unreliable due to neglect, sexual, physical, or emotional abuse. Due to multiple ED presentations, patients with personality disorders are often coined as “frequent flyers” and in so doing induce a sense of complacency in the evaluator. Accurate assessment of the patient’s medical status can be difficult when an opinion of the patient has already been formed. Studies have shown that physical examinations on psychiatric patients are often incomplete. It is easy to view the patient strictly as a psychiatric patient and dismiss other possible medical causes for their ED presentation, and this bias can be intensified with multiple visits.

Although patients with personality disorders may present frequently to the ED their reasons for visiting may vary and include both psychiatric and medical complaints. Inadequate history and physical examinations are the leading causes of missed diagnoses of medical conditions masquerading as psychiatric conditions. Medical clearance of a psychiatric patient involves ruling out underlying organic illness, infectious cause, intoxication, or toxidrome as well as assessing an accurate set of vital signs. When the screening evaluation and examination is complete, the patient can then be medically cleared for disposition from the ED for further psychiatric evaluation.

## KEY POINTS

- When interacting with patients with personality disorders, be direct,

professional, and empathetic, and remember not to react emotionally to dramatic or inappropriate behavior.

- Be aware that patients with personality disorders frequently have multiple comorbid conditions including substance abuse, anxiety, and depression.
- Document your interactions with the patient thoroughly as the record may help to provide insights and clarity later when managing the patient's condition.
- Since patients with personality disorders may present to the ED for multiple reasons, it is important to perform thorough history and physical examinations to rule out organic illnesses that may masquerade as psychiatric complaints.

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# DON'T IGNORE AFFECTIVE DISORDERS!

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TRENT R. MALCOLM, MD, MS

## CASE

*A 53-year-old woman with a history of hypertension, dyslipidemia, and hypothyroidism presents with abdominal pain. She reports several weeks of dull, generalized abdominal pain that does not have any clear aggravating or alleviating factors. On exam, she is noted to have a flat affect but otherwise unremarkable examination. After further questioning, she endorses low mood, difficulty sleeping, and loss of interest in her normal activities. When asked if she ever has thoughts of hurting herself, she begins to cry.*

Mood disorders, including major depression and bipolar disorder, are disproportionately represented in the emergency department. The lifetime prevalences of depression and bipolar disorder in the general population are ~ 13% and 1%, yet these conditions are present in as many as 25% to 30% and 7% of all emergency department patients, respectively. Patients with psychiatric illness present unique challenges for the emergency provider and carry increased risk of poor outcomes. For example, mood disorders frequently manifest with somatic complaints that cause diagnostic challenges. Worryingly, psychiatric illness may mask underlying medical illness resulting in serious conditions going overlooked and undiagnosed. In addition, patients with mood disorders have significantly elevated risk of suicide, have high rates of substance abuse, demonstrate lower adherence

with management of chronic illnesses, and utilize emergency health care services at significantly higher rates. Although there may be a cognitive tendency to deemphasize the emergency care of patients with mood disorders, extra care should be taken to address the medical, psychological, and social needs of this at-risk patient population.

Classic symptoms of depression include low mood, loss of interest, and thoughts of hopelessness or guilt. When such psychosocial symptoms are present, clinical suspicion for major depression is readily piqued. However, the presentation of major depression is often insidious. Somatic complaints dominate in up to two-thirds of patients, leading to delayed and missed diagnoses. Common somatic complaints include fatigue, diffuse body aches, abdominal pain, chest pain, dizziness, loss of appetite, and sleep disturbance. In patients with unexplained complaints following an appropriate medical evaluation, consider undiagnosed depression in the differential diagnosis. A few simple screening questions may lead to an unexpected diagnosis and facilitate linkage to appropriate psychiatric care.

Conversely, a disturbing number of medical conditions may masquerade as depression. Don't be fooled into thinking that low mood and loss of interest are always psychiatric in origin. Common causes of reversible or secondary depressive symptoms in the ED include endocrine abnormalities (e.g., hypothyroidism, Cushing syndrome), medication effects (e.g., antihypertensives, hormone therapy), nutritional deficiencies (e.g., B<sub>12</sub>, thiamine), toxic ingestions, infections, and CNS disorders. In patients presenting with mood symptoms without prior history of depression, it is essential to undertake a thorough evaluation for potentially reversible causes. Key components include a detailed medical history, medication history, and physical examination and laboratory studies. A broad differential diagnosis and a keen diagnostic sense are needed to identify potentially serious medical illnesses and avoid unnecessary psych referrals for patients with organic causes of depression.

All patients with mood disorders or evidence of depression should be screened for suicidal ideation. Mood disorders are significant risk factors for suicide; the lifetime risk of suicide is roughly eight times higher in patients with mood disorders than the general public. Suicide is one of the leading causes of life-years lost and a major public health problem. In addition, emergency providers may be in a unique position to identify and intervene in a timely fashion given the frequency of ED visits for patients with mood disorders. Patients that screen positive for thoughts of suicide should undergo comprehensive suicidal risk assessment (see [Chapter 215](#), Ask about Suicide Risk) and treated accordingly.

Evaluation of patients with mood disorders in the emergency department ultimately provides an opportunity to connect high-risk individuals to appropriate care. Patients with mood disorders or depressive symptoms should receive close follow-up by their primary care physician or psychiatrist. Sadly, many of these patients do not have access to regular follow-up. If available, utilize case management and/or social work resources to help establish care for these patients. Finally, emergency providers are cautioned against initiating antidepressive medications for treatment of mood disorders. Given the risk of increased suicidality immediately after initiation of certain antidepressants, the decision to start such medications is best left to primary care or psychiatric providers that are better able to closely monitor the effects of treatment.

## KEY POINTS

- Always consider depression in patients with unexplained somatic symptoms.
- Conversely, be certain to rule out organic illness in patients with depressive symptoms.
- *All* patients with depressive symptoms should be screened for suicidal ideation; be sure to complete a detailed risk assessment when appropriate.
- Think twice before prescribing antidepressant medications in the emergency department.
- Refer all patients with evidence of a mood disorder to their primary care provider or to psychiatry for close follow-up.

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# DRUG-SEEKING BEHAVIOR IN THE EMERGENCY DEPARTMENT

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**MICHAEL WOLFE PIERCE, MD**

There is undeniably an epidemic of prescription drug misuse and abuse in the United States. Death from drug overdose (most commonly prescription opioids) recently surpassed motor vehicle collisions as the leading cause of accidental death nationwide. The effects of this widespread problem are clearly seen in emergency departments, where overdose is a common presenting complaint, but unfortunately, the same facilities are also a common source of the offending drugs. Treating pain is a core component of emergency medical practice. By many estimates, a majority of emergency department visits are motivated at least in part by the desire for pain relief. While the decision to treat pain from cancer or acute pain syndromes is uncontroversial, the treatment of patients with chronic pain or those without any objectively identifiable pain source remains fraught with potential complications.

Appropriately differentiating patients primarily seeking drugs from those with legitimate therapeutic need is an admirable goal, but not one that is presently achievable in a busy emergency department. Still, physicians are tasked with the responsibility not only to appropriately treat their patients' pain and suffering but also to protect them from the ill effects of unnecessary treatment. Medication side effects, addictions, and their sequelae harm both those who misuse for recreation and those who use for relief of chronic pain or distress. Indeed, overt addiction represents only a small component of the overall picture of problematic drug use; a much more common manifestation is a subtle dependence, which is not readily apparent to the physician or even the patient.

Central to the problem from the prescriber's perspective is the lack of

objective means to verify a patient's stated presence or severity of pain. There are some classical behaviors that have been shown to be associated with drug-seeking behavior in the emergency department, but none have been shown to be particularly sensitive or specific. These include patients with impressive lists of allergies to all but their drug(s) of choice, patients stating that any nonopioid or benzodiazepine medications are ineffective (preoccupation with drug class), patients requesting medication by name, and patients stating prior prescription(s) were lost or stolen. Similarly, there are several classically described chief complaints including headache, dental pain, and abdominal/pelvic pain, which have been associated with drug seeking. Of course, any of these elements of a patient's history can be present for someone in dire need of analgesia, but their presence warrants the application of particularly careful clinical decision making.

When assessing whether a patient is drug seeking, it is critical to remember that clinician bias, especially with respect to socioeconomic, educational, or racial background, age, and gender significantly impact treatment decisions on the management of pain. Introspection and frequent review of one's own decision making are enormously important in these situations, and giving patients the benefit of the doubt with respect to the voracity of their pain is generally the best approach. Anyone driven to the point of asking for pain relief in the emergency department is having a terrible day one way or another and is worthy of significant empathy. Similarly, patients with known drug seeking and even disruptive behavior in the emergency department develop life-threatening organic pathology, and this possibility must be taken seriously on every presentation.

The necessary but difficult course of declining to prescribe when faced with patients who specifically request drugs when they are not indicated is challenging, but can be accomplished with compassion. Ideally, this is done by acknowledging the patient's distress while explaining that the requested medications are likely to do them more harm than good. This is an accurate but particularly unwelcome recommendation for some patients when discussing their use of short-acting opioids for chronic pain. Continued demands for medication once the clinician's reasoning has been made clear, and the patient has acknowledged that they are requesting a potentially harmful treatment, warrants a discussion of psychological and physiological dependence. Unfortunately, even the most compassionate and well-reasoned refusal to prescribe may not be well received and may even lead to behavioral escalation. The clinician's approach must be tailored to the patient as well as their specific presentation and, even in the best case, may not be successful until it has been reinforced by multiple physicians.

Patients with chronic pain and/or drug dependency are ideally managed

in the context of a longitudinal primary care relationship as the often hurried and impersonal environment of the emergency department presents additional challenges. That said, the emergency department can also be an ideal setting for the initial identification and first steps of management for drug abuse. Patients at the highest risk for abuse and addiction are those who frequently present to the emergency department with the goal of obtaining opioids (frequently in the setting of subtherapeutic chronic pain management). Those with pre-existing psychological and or psychosocial dysfunction are particularly vulnerable. This subset of patients is even less likely to have a relationship with a primary care physician, which makes this point of contact with health care all the more essential. Emergency department patients with drug dependence are often seeking help in one form or another and have often presented as the result of a crisis directly or indirectly related to their use or supply of the offending drug. In this setting, patients may be more amenable to the suggestion of behavioral change and referral to outpatient treatment programs. Specifically, those with known opioid dependency who present exhibiting symptoms of withdrawal are best managed with lower-risk opioids with less euphoric properties (i.e., buprenorphine or methadone) and should be supported in seeking additional addiction therapy.

## KEY POINTS

- There is an epidemic of prescription drug misuse and abuse with devastating individual and societal consequences.
- Clinicians must always weigh the importance of treating pain against the potential harms to each patient. It is crucial to consider one's own biases in the decision-making process.
- Declining requests for unnecessary medications should be done with compassion; anyone asking is having a terrible day.
- Presentation to the emergency department is an excellent time for intervention on drug abuse (prescription or otherwise).

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# **ANXIETY IN THE EMERGENCY DEPARTMENT**

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**SACHIN MOONAT, MD, PhD**

A visit to emergency department (ED) is a nerve-wracking experience for a patient, an individual with unexpected health concerns with an uncertain outcome in an unfamiliar environment. As emergency physicians, we can quickly get a sense of the emotional distress that a patient may be experiencing. We can see the panic in our patient's facial expressions and hear it in their voices. We understand that anxiety contributes substantially to a patient's discomfort and dysphoria. Although nearly 75% of patients report some degree of anxiety when presenting to the ED (with about 25% reporting severe anxiety), these concerns are addressed in <5% of patients.

## **THE ANXIOUS STATE**

In certain situations, the anxious state can be biologically beneficial. The limbic system, in particular the amygdala, facilitates autonomic arousal under conditions of uncertainty or distress. The increase in sympathetic tone contributes to a state of readiness with activation of the “fight-or-flight” response. Pupils dilate, cardiac output increases, reflexes quicken, and hypervigilance ensues. Although these physiologic reactions may provide benefit during certain stressful situations, in practice, the anxious ED patient reports uneasiness, distress, and feelings of impending doom. These feelings are frequently observed in the ED as somatic complaints such as chest pain, palpitations, dyspnea, gastrointestinal discomfort, diaphoresis, light-headedness, weakness, and fatigue. It is important to note that patients are not generally aware that these symptoms may be associated with anxiety. The pain threshold is also reduced by the anxious state, so patients may

complain of severe pain that seems incongruent with their physical exam.

## EVALUATING THE ANXIOUS PATIENT IN THE ED

The symptoms of anxiety may be due to the chaotic ED environment (“white coat” anxiety), a serious underlying medical condition, drug or alcohol intoxication or withdrawal, or a primary anxiety disorder. More than one condition may be present concurrently. Although it may be tempting to diagnose primary anxiety to arrive at a quick disposition, it is important to address the patient’s symptoms seriously. This helps to establish a relationship of trust and ensures no medical conditions will be overlooked.

Engaging the anxious patient with open-ended questions and listening calmly can help to limit distress and may actually be therapeutic. Determining if environmental factors or life stressors have triggered the episode of anxiety can help to discern exogenous and endogenous anxiety. Exogenous anxiety in the context of acute environmental stress is frequently amenable to reassurance. These patients will benefit from a discussion with the social worker or referral to a mental health professional. On the other hand, primary anxiety disorders are endogenous, having genetic and long-term environmental contributions, such as chronic stress. Exacerbations often occur without an identifiable trigger and patients may have a history of recurrent attacks. If a primary anxiety disorder is suspected, a referral for psychiatric evaluation should be suggested. The anxiety spectrum covers a variety of specific diagnoses. For the purposes of this text, and the emergency provider, it is more important to differentiate between primary and secondary causes of anxiety than to make a specific psychiatric diagnosis.

Suicidal or homicidal ideation must be addressed in any severely anxious patients, leading to consideration for emergent psychiatric evaluation and admission. It is important to assess drug and alcohol use in all anxious patients. Substance use and withdrawal, in particular alcohol withdrawal, are common causes of anxiety. Stimulants, including caffeine and nicotine, can worsen anxiety and should be avoided by these patients.

Even if a psychiatric etiology is suspected, the workup of anxiety must address the possibility of an underlying medical condition. Missing a myocardial infarction or pulmonary embolism in a patient discharged with a diagnosis of anxiety would be disastrous. Somatic complaints need to be taken seriously and should guide further workup. Certain cardiovascular, respiratory, endocrine, neurologic, and toxicologic pathologies can cause anxiety as a primary symptom (see [Table 219.1](#)). The anxious patient may answer yes to almost the entire review of systems checklist. This should lead

the provider to consider a workup for common medical etiologies of anxiety, as the history cannot be trusted reliably. Although anxiety may cause tachycardia, tachypnea, and hypertension, primary medical conditions should be ruled out when abnormal vital signs are present.

**TABLE 219.1 MEDICAL CONDITIONS WITH ANXIETY AS A COMMON SYMPTOM**

|                       | <b>CONDITIONS</b>  | <b>TESTS TO CONSIDER</b>                                      |
|-----------------------|--|---|
| <i>Cardiovascular</i> | MI, angina, ADHF, dysrhythmia, mitral valve prolapse   | ECG, cardiac enzymes, stress testing, p-BNP, echocardiography |
| <i>Pulmonary</i>      | PE, asthma, COPD, spontaneous pneumothorax   | CXR, D-dimer, VQ scan, CTA chest                              |
| <i>Endocrine</i>      | Hyper-/hypothyroidism, hypoparathyroidism, hyper-/hypoglycemia, pheochromocytoma, hypercortisolism | Glucose, TSH, free T4, calcium, PTH, cortisol                 |
| <i>Neurologic</i>     | CVA, TIA, seizure, epilepsy, multiple sclerosis, Huntington, Alzheimer, Parkinson, TBI, concussion | CT head, MRI, EEG, CSF studies                                |

From Craven P, et al. Patient anxiety may influence the efficacy of ED pain management. *Am J Emerg Med.* 2013;31(2):313–318.

## TREATING THE ANXIOUS PATIENT IN THE ED

Regardless of the cause, providers should attempt to alleviate symptoms of anxiety in the ED. The therapeutic process may be initiated with calm reassurance during the patient interview. In some cases, anxiolytics such as diphenhydramine or, in select cases, benzodiazepines can be considered for patients in continued distress. Appropriate management of the symptoms of anxiety has been shown to reduce morbidity and to increase patient satisfaction. In fact, studies among postmyocardial infarction patients have shown that use of anxiolytics in appropriate patients reduces mortality. Providers are frequently cited stating that they do not want to “mask” symptoms of an underlying medical condition by administering benzodiazepines. This is not an appropriate reason to leave a patient in continued distress. Treatment of anxiety is generally achieved through low-dose oral benzodiazepines. Patients do not usually require intravenous or intramuscular doses, which are often needed for the agitated ED patient. On the contrary, use of low-dose benzodiazepines may calm the anxious patient enough to get a more reliable history and exam and may reduce the amount

of analgesia required to treat the patient's pain. Make sure to address the patient's medication list prior to initiation of anxiolytics. Caution should always be used when administering benzodiazepines as a paradoxical reaction can occur in certain populations resulting in excessive movement, emotional release, and increased talkativeness. Benzodiazepines may be less effective in patients that have developed a tolerance through chronic use. Be careful when considering prescribing benzodiazepines for outpatient management. It would be advisable to restrict anxiolytics to a limited prescription for breakthrough anxiety, in order to avoid complications of tolerance and dependence. Patients requiring ongoing pharmacotherapy should be advised to speak with their primary care physician or a psychiatrist for further management.

## KEY POINTS

- Calmly listen to the complaints of anxious patients; your interview can be therapeutic!
- Take all somatic complaints seriously and rule out any potential medical causes of anxiety.
- Consider toxicologic causes of anxiety and counsel patients to avoid anxiogenic substances such as caffeine, nicotine, alcohol, and illicit drugs.
- Assess for suicidal or homicidal ideation in the severely anxious patient.
- Provide reassurance and appropriate referral to patients with primary exogenous and endogenous anxiety.
- Treatment of anxiety with anxiolytics helps to reduce distress and can help reliably assess the anxious patient.

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# **ADDRESS IT IN THE ED, SUBSTANCE ABUSE IN THE EMERGENCY DEPARTMENT**

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**SACHIN MOONAT, MD, PhD**

## **REMEMBER TO ADDRESS CHRONIC SUBSTANCE ABUSE ISSUES IN ED PATIENTS**

As emergency providers, our role in the care of patients with substance abuse issues frequently amounts to stabilization and supportive care during episodes of intoxication, overdose, or withdrawal. We are continually faced with challenging patients that require benzodiazepines for alcohol withdrawal or naloxone for opioid overdose. While we do an excellent job of treating patients' emergent needs, we often forget to address their chronic condition of substance abuse. The National Institute of Drug Abuse estimates that nearly 25 million Americans aged 12 and above have used drugs of abuse within the past month. On a global scale, the World Health Organization estimates that use of alcohol, tobacco, and illicit drugs account for over twelve percent of all deaths. Although only a fraction of these patients may present to the emergency department (ED) due to consequences of drug use, the emergency physician is in a unique position to initiate screening and interventional strategies aimed toward treatment and harm reduction.

## **SCREENING IN THE ED**

Given the high prevalence of drug and alcohol use, efforts should be taken to

assess substance abuse in all patients. It is important to take efforts to avoid sounding accusatory or blaming the patient for consumption behaviors. Substance abuse is a disease like any other and not the fault of the individual. In patients presenting with intoxication or overdose of a specific agent, coingestions and polysubstance abuse should be addressed. Screening for substance abuse should include amount and frequency of consumption. This can be addressed by asking patients about the number of days they use per week, the amount consumed per episode, and amount consumed per week. Screening questions should also be directed toward the social, health, and economic consequences of a patient's substance use. Clinically, significant drug and alcohol abuse may be difficult to accurately assess due to the reticence of patients to divulge this information. Because of this, effective screening tools, which address the patient's own view of their alcohol or drug consumption, have been developed. Two worthwhile screening tools to consider are the CAGE-AID and CRAFFT questionnaires (see [Table 220.1](#)). The CAGE-AID questionnaire is used to screen for both substance and alcohol abuse. The CRAFFT questionnaire is primarily aimed at identifying risky substance abuse behaviors among adolescents. Positive screening using formal screening tools or high clinical suspicion of substance abuse should prompt the emergency physician to employ a brief intervention.

**TABLE 220.1 CAGE-AID AND CRAFFT SCREENING QUESTIONNAIRES**

**CAGE-AID Questions**

- Have you ever felt should Cut down your drinking or drug use?
- Have you felt Annoyed by people criticizing your drinking or drug use?
- Have you ever felt bad or Guilty about your drinking or drug use?
- Have you ever had a drink or used drugs in the morning to steady your nerves or get rid of a hangover (Eye opener)?

**CRAFFT Questions**

- Have you ever been in a Car being driven by someone (including yourself) who had been using alcohol or drugs?
- Do you ever use alcohol or drugs to Relax, feel better about yourself, or fit in?
- Do you ever use alcohol or drugs when you are Alone?
- Do you ever Forget things that you did while using alcohol or drugs?
- Do your Family or Friends ever tell you to cut down your alcohol or drug use?
- Have you ever gotten into Trouble while using alcohol or drugs?

Patients with positive findings on 2 or more questions may have a clinically significant alcohol use disorder and warrant further examination and counseling.

## **INTERVENTION IN THE ED**

A frequently cited barrier to behavioral intervention in the ED is the lack of time to address substance abuse issues. To this end, patients are frequently discharged, admitted, or referred to outside resources without substance abuse issues being discussed directly with the emergency physician. It has been suggested that a brief intervention of 10 to 15 minutes may be successful in motivating substance-dependent patients to seek or continue treatment. Of note, episodes that highlight the social, legal, and health consequences of substance use, such as those that bring patients into the ED, may be the most effective time to initiate intervention. The “brief negotiated interview” is currently believed to be the most rapid and efficacious technique to increase patient enrollment in outpatient substance abuse programs. In order to help patients make the personal decision to seek substance abuse treatment, the motivational interviewing technique relies on four key strategies: (1) establishing rapport, (2) providing feedback on consequences of substance use, (3) enhancing motivation to seek assistance, and (4) negotiating a goal and course for follow-up. Although it is beyond the scope of this chapter to provide training into motivational interviewing, numerous resources, online videos, and courses are available to the ED provider.

## **REFERRAL FOR TREATMENT**

The conclusion of the brief negotiated interview should create a bridge to providing treatment resources for a patient. EDs should have a list of resources available to patients, and suggestions should be made based on the individual needs of a patient. Social work and case management may be employed to assess financial or insurance requirements to identify feasible treatment options for an individual. Most jurisdictions have free community-run outpatient detoxification and treatment programs, including Alcoholics and Narcotics Anonymous. Yet, many patients may not be ready to commit to these steps at the time of interview. These patients should be recommended close follow-up with their primary care physician. If they do not have one, attempts should be made to establish a primary care physician. In less motivated patients, the ED physician may be able to promote follow-up by placing greater emphasis on continued health maintenance rather than on substance abuse counseling.

## **HARM REDUCTION**

It is worth noting that numerous studies have found that brief intervention in the ED is ineffective with regard to an outcome of abstinence from substance use. Yet, from a public health standpoint, it is important to consider that this may not be the most useful primary outcome to study. As ED providers, we are exposed to the acute health consequences of substance abuse. Interventions aimed at reducing these consequences are at least as important as reducing substance abuse. It is important to remember that we can promote safer drug use practices leading to reductions in driving while intoxicated, greater use of needle-exchange programs, and awareness of overdose practices. Recently, the introduction of naloxone prescriptions and training programs has made the treatment of opiate overdose available to drug users and their friends and families. As physicians on the front lines, we should strive to reduce the stigma toward methods of harm reduction and promote utilization of such services. Although the issue of substance abuse is complex and seemingly intractable, numerous interventions and strategies can be employed in the ED to reduce negative consequences for patients with substance abuse and promote safe practices.

## KEY POINTS

- Avoid accusatory statements regarding consumption behaviors and establish rapport with patients prior to initiating a conversation regarding substance abuse.
- Any suspicion of drug or alcohol use should prompt providers to *screen* for clinically significant substance use disorders.
- Attempts should be made to *intervene* with a brief negotiated interview to initiate a conversation about substance abuse.
- Remember to provide *follow-up* with a referral to a treatment program or, if the patient is unwilling, health-focused outpatient care.
- Provide resources and prescriptions to *reduce harm*, even if a patient is unwilling to abstain from or reduce substance consumption.

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## SECTION XVI

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# GENITOURINARY

# **DON'T LET DIALYSIS DISEQUILIBRIUM SYNDROME CATCH YOU OFF-BALANCE**

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**CARMEN WOLFE, MD**

Emergency providers may be called upon to evaluate patients who develop neurologic symptoms during hemodialysis. An uncommon, though important and often missed, cause of this presentation is dialysis disequilibrium syndrome (DDS). DDS is a rare syndrome encompassing a variety of neurologic complaints, and recognition of this potentially life-threatening condition is the key to ensuring immediate targeted treatment.

Presentation of DDS may occur during dialysis or up to 24 hours after a dialysis session. Minor symptoms include headache, nausea, dizziness, or muscle cramps. More serious presentations range from altered mental status and seizures to coma and death.

The underlying mechanism leading to DDS is the acute onset of cerebral edema. While the exact etiology is still debated, the leading theory for the mechanism of edema is a reverse osmotic shift. As urea is rapidly removed from the peripheral circulation during hemodialysis, a lag in the shift of urea out of brain tissue leads to the creation of an osmotic gradient and, subsequently, cerebral edema.

While DDS can develop in any patient undergoing hemodialysis, specific risk factors make this diagnosis more likely. Severe uremia, first-time dialysis, severe metabolic acidosis, chronic kidney disease (as opposed to acute onset), extremes of age (both elderly and pediatric patients), and underlying neurologic disease can all increase the risk for DDS. Additionally, any condition that increases the permeability of the blood-brain



barrier such as sepsis, meningitis, or vasculitis may also lead to an increased risk.

DDS should be considered a diagnosis of exclusion. Clinicians should first pursue a workup of other more common causes of altered mental status prior to arriving at this diagnosis. This begins with vital signs; hypotension during and after dialysis may lead to watershed cerebral ischemia. Standard laboratory testing may reveal derangements in glucose, sodium, or calcium that may account for the patient's symptoms. Neuroimaging should be performed to rule out stroke or intracranial hemorrhage, which is much more common in patients on dialysis due to their acquired coagulopathy. An infectious workup, including chest x-ray, urine, and blood cultures, is also appropriate if there is any question of sepsis. Finally, an evaluation of the patient's medications may reveal a drug-induced encephalopathy as a potential cause.

The treatment of DDS begins with supportive care as the initial resuscitation is directed at securing the ABCs and ruling out the correctable abnormalities listed above. If dialysis is in progress, it should be stopped immediately. The main targeted treatment strategy for DDS involves the management of cerebral edema. Intravenous mannitol, hypertonic saline, and mechanical hyperventilation may all be utilized to try to lower intracranial pressure (ICP).

DDS is becoming increasingly uncommon as nephrologists have altered their algorithms for beginning dialysis in an attempt to prevent this potentially fatal condition. Nevertheless, DDS still occurs and often goes unrecognized until symptoms are severe and irreversible. Because the diagnosis of DDS carries such a poor prognosis, it is important to consider in any hemodialysis patient in whom another more obvious, reversible cause of neurologic symptoms is not apparent.

## KEY POINTS

- Consider the diagnosis of DDS when a patient undergoing hemodialysis presents with any neurologic complaint.
- The primary pathology of DDS is acute-onset cerebral edema.
- DDS is a diagnosis of exclusion—rule out other life-threatening causes of altered mental status prior to assuming DDS.
- Treatment for DDS includes standard treatments for elevated ICP such as mannitol, hypertonic saline, and hyperventilation; however, the mortality remains high.

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# FOURNIER GANGRENE: A LETHAL INFECTION YOU CAN'T SIT ON!

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SHOMA DESAI, MD

With an average mortality rate of 20% to 30%, Fournier gangrene is a true emergency requiring prompt diagnosis and aggressive treatment. Named after Jean-Alfred Fournier, a French venereologist of the late 1800s, it is defined as a fulminant, necrotizing fasciitis of the perineal, genital, and/or perianal regions. Fournier gangrene is an uncommonly encountered disease with potentially devastating effects. Classically, sources of this soft tissue infection include the gastrointestinal tract (e.g., perianal abscess, colonic perforation, malignancy), the genitourinary tract (e.g., indwelling catheters, urethral calculi), and local cutaneous disease (e.g., hidradenitis suppurativa, skin ulcers). Local trauma, in the form of piercings, penile implants, drug injections, and rectal foreign bodies, is a recognized factor in some cases. Fournier gangrene most frequently targets the scrotum and penis or anorectal region; however, advanced cases may involve the anterior abdominal wall, chest wall, or thighs. Classically, this necrotizing soft tissue infection spares deeper muscular structures.

Since survival hinges on early diagnosis, the clinician must be aware of its range of clinical symptoms and signs. A classic presentation of Fournier gangrene involves a diabetic male with rapidly progressing perineal pain, redness, and swelling. One may note crepitus upon palpation (due to bacterial gas formation) and pain out of proportion to examination. In more advanced stages, a dusky appearance and frankly necrotic tissue will appear. However, less classic patients or those with atypical presentations can be missed.

In rare cases, Fournier gangrene is seen in women and children. Gynecologic sources of infection include Bartholin gland abscess, septic

abortions, and pelvic surgery. Pediatric variants may arise from circumcision, omphalitis, or a strangulated inguinal hernia. Also, while the majority of these patients are diabetic (up to 70%), several chronic conditions that impair immunity have been described as risk factors for this disease. These comorbidities include cirrhosis, alcohol abuse, HIV, systemic lupus erythematosus, malignancy (such as leukemia), and chronic steroid use.

Most importantly, not all cases of Fournier gangrene are clinically overt. Early in the course, an inspection of the skin may be relatively benign or even normal, belying severe damage to deeper tissue. In addition, this necrotizing soft tissue infection can fool the clinician with an insidious onset and slow progression. In these patients, systemic signs may tip the emergency provider off. Patients frequently present with systemic inflammatory response syndrome (SIRS), vomiting, lethargy, or, in advanced cases, septic shock with multiorgan failure. Typically, Fournier gangrene is associated with pain, but late presenting infections may have relatively little pain due to destruction of nervous tissue.

Because the clinical presentation does not always reveal the true extent of the infection, diagnostic imaging may be necessary to solidify the diagnosis. Plain radiography may show air along the fascial planes, but its absence does not exclude the diagnosis. Ultrasound may show an edematous scrotal wall with gas artifact and is useful to rule out other causes of acute scrotal pain, such as testicular torsion. Computed tomography (CT) and, less commonly, magnetic resonance imaging (MRI) are obtained to look for subcutaneous air, thickened fascia, and fat stranding. Of note, the testes are classically spared due to their direct blood supply from the aorta via the testicular arteries; therefore, any testicular involvement (i.e., orchitis) points to a retroperitoneal or intra-abdominal source of infection. This may be delineated by advanced imaging (i.e., CT or MRI). Because early diagnosis is critical to early management, it is important that the process of obtaining advanced imaging never delays surgical consultation.

Once the diagnosis of Fournier gangrene is entertained, an aggressive management plan should be instituted. Hemodynamic stabilization and early antibiotics are key components of treatment. Since the pathogenesis of this necrotizing fasciitis is polymicrobial, involving both aerobic and anaerobic bacteria, broad-spectrum antibiotics should be administered. Bacterial culprits include coliforms (most commonly *Escherichia coli*), *Streptococci*, *Staphylococci*, *Clostridia*, *Bacteroides*, and *Pseudomonas* spp. Rarely, fungus may be involved, such as *Candida albicans*. The standard regimen involves triple therapy, usually a combination of clindamycin, a beta-lactam antimicrobial, and metronidazole. Vancomycin may be added for extended gram-positive coverage. Amphotericin B may be used in the case of fungal

infection. Early aggressive treatment, in the form of surgical debridement, is associated with a reduced mortality rate. A multidisciplinary approach, involving emergent consultation with general surgery and urology (or gynecology), is optimal. In surgery, pus described vividly as “dirty dishwater fluid” may be seen and necrotic tissue must be removed. Patients may require several surgical procedures to control the infection; one study reported a whopping 3.5 surgical procedures per patient on average.

The consequences of delayed diagnosis and/or surgical management are numerous, debilitating, and often life threatening. In the short term, death may result from diabetic ketoacidosis, septic shock, coagulopathy, acute renal failure, or multiorgan failure. Those who survive may suffer from lifelong pain, sexual dysfunction, bowel incontinence, disfiguring scars, and/or lymphedema.

## KEY POINTS

- Fournier gangrene may present atypically—pay attention to systemic signs, feel for crepitus, and don’t expect pain out of proportion to examination.
- Once suspected, resuscitative efforts, early antibiotics, and emergent surgical consultation are critical to saving the patient’s life.

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## TESTICULAR TORSION TRICKERY

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**SOLOMON BEHAR, MD**

Testicular torsion occurs when the testis twists on itself, cutting off arterial blood supply to the affected testicle and resulting in a dead testicle if prompt intervention is not instituted. There are two age peaks of testicular torsion—one in infancy and another in early adolescence. Normally, the testicle is fixed to the tunica vaginalis posteriorly and superiorly. Patients with congenital anatomic variants such as the “bell clapper” deformity, where the testicle is not attached posteriorly but instead 360 degrees around the tunica vaginalis superiorly, are at increased risk for testicular torsion. Testicular torsion is the cause of acute scrotal pain in 15% to 30% of cases.

Symptoms of testicular torsion include sudden onset of pain, vomiting, redness, and swelling of the affected testicle. On examination, typical findings include erythema, swelling, and a change in position of the testicle from a vertical to horizontal lie. An absent cremasteric reflex may occur during testicular torsion, but its presence does not rule out the diagnosis of torsion. Be wary of the adolescent male who complains of “abdominal pain” and always include a testicular examination in your evaluation of these sometimes shy patients who may not readily admit to having pain in their genital area. In one retrospective study looking at 84 cases of testicular torsion, 9 boys (11%) presented with abdominal pain and vomiting. Six of those cases were missed and had no documented testicular examination, and three cases were missed despite a documented genitourinary (GU) exam. Remember as well that patients with a history of testicular trauma are at increased risk for torsion.

Patients who have testicular torsion often have a history of testicular pain, having a pattern of “torsion/detorsion.” Do not forego evaluation by ultrasound (US) of a patient who presents with worsening testicular pain simply because they had a recent “negative ultrasound.” Return precautions

in the patient who is pain free and has a reassuring US should include a documented discussion of the torsion/detorsion phenomenon.

Another high-risk group is the nonverbal or preverbal patient who presents with acute pain or crying of unknown origin. Always do a GU examination to look for the cardinal signs of torsion in the male infant presenting with crying as a chief complaint. If the testes are not easily palpated in the infant's scrotum, that heightens concern. Patients with undescended testicles are at increased risk for testicular torsion. With no obvious outward physical examination findings to suggest torsion of an intra-abdominal testicle, one must remember to consider it. US findings of torsion of the intra-abdominal testicle can also be challenging to interpret compared with that of testis in the scrotal sac. If clinical suspicion is high, surgical evaluation should still be pursued regardless of US results.

Treatment consists of detorsion, either manually or surgically, as soon as possible. Salvage rates of torsed testicles plummet after 12 hours of symptoms, and ideally, torsion should be definitively fixed prior to 6 hours of symptoms. Manual detorsion should be attempted if surgical resources are not available in a timely fashion. When performing manual detorsion, classic teaching is rotation of the testicle outward in a medial to lateral direction (like you are "opening a book"). However, one retrospective study by Sessions et al. showed that up to one-third of cases were rotated in the opposite direction, so clinical symptom improvement and restoration of blood flow on US (if available) should be noted when performing manual detorsion. Remember, the testis may be twisted more than 360 degrees, so continue detorsion until the patient expresses pain relief or a bedside US shows return of blood flow to the affected testicle. Even after manual detorsion, exploratory surgery must be done as soon as possible, as residual torsion may exist in up to 1/3 of patients.

## KEY POINTS

- Consider testicular torsion in a preverbal or nonverbal patient who presents with crying as the chief complaint.
- Do not hesitate to order an US in a patient with history of testicle pain and a normal prior US—they may be experiencing torsion/detorsion.
- To maximize salvage of the torsed testicle, repair should ideally occur prior to 6 hours from onset of pain.
- Manual detorsion should be attempted if a prolonged time to definitive repair is expected.

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# HEMODIALYSIS: WHO NEEDS IT NOW?

**MICHAEL LEVINE, MD**

Emergency practitioners frequently encounter patients with renal failure, both chronic and acute. Many patients with acute kidney injury (AKI) are treated without hemodialysis, at least initially, while other patients will require emergent hemodialysis.

Patients with chronic renal failure who miss their regular dialysis appointment may present with volume overload or hyperkalemia and require dialysis. In such cases, if the patient is not significantly hypoxic, and is able to be stabilized medically (e.g., nitroglycerin, diuretics and oxygen for volume overload, or Kayexalate, albuterol, glucose/insulin for hyperkalemia), the patient may be able to wait until routine dialysis is available (i.e., during regular clinic hours). However, if medical management is ineffective at stabilizing the patient, dialysis must be performed emergently.

In general, hemodialysis can be used in the emergent setting to remove toxins or to correct electrolytes and/or volume status. The generally accepted indications for emergent hemodialysis include pulmonary edema, hyperkalemia, or metabolic acidosis refractory to medical management, uremia, and removal of toxins. Common toxins that are amenable to removal with high-flux hemodialysis are listed in [Table 224.1](#).

**TABLE 224.1 COMMON TOXINS THAT MAY BE AMENABLE TO HEMODIALYSIS<sup>A</sup>**

## Alcohols

- Ethylene glycol
- Methanol
- Propylene glycol

Aspirin (salicylates)

Bromides

Lithium

Theophylline

<sup>a</sup> This list is not inclusive and should not serve as a substitute for consultation from a regional poison control center or toxicologist.

Inappropriate utilization of hemodialysis is a common pitfall for emergency practitioners. Patients with volume overload or electrolyte disturbances may warrant a trial of medical management before deciding on the need for emergent hemodialysis. However, once it is apparent that medical treatment is not working as efficiently as required by the situation, substantial delays in implementing dialysis in such settings can have catastrophic results.

In general, the decision to institute hemodialysis for toxin removal should be based on the clinical syndrome, not strictly on a particular serum or whole blood level. Don't base the decision to dialyze exclusively on the serum level for agents such as lithium and aspirin. These values are general estimates and should not be viewed as the exclusive indications for dialysis. For lithium, thresholds of 4 mEq/L in acute toxicity or >2.5 mEq/L in chronic toxicity are typically set as indications for dialysis. However, most toxicologists believe that neurotoxicity, independent of level, is the primary indication for dialysis in lithium toxicity.

Some advocate dialysis for salicylate toxicity if the salicylate concentration exceeds 100 mg/dL in acute toxicity or 60 mg/dL in chronic toxicity, regardless of other findings. Other, more widely, accepted indications for hemodialysis in salicylate toxicity include failure of medical management, as well neurological compromise attributed to the salicylate toxicity, pulmonary edema, or renal failure that would preclude the administration of large volumes of intravenous fluid, as well as refractory acid-base disturbances. A patient who is rapidly improving with medical

management who has no other indications for dialysis may have a salicylate concentration exceeding 100 mg/dL and not warrant hemodialysis, assuming there is rapid improvement in the clinical syndrome with accompanying falling serum concentrations. If the patient is not rapidly improving and the salicylate concentration exceeds 100 mg/dL in acute toxicity, emergent hemodialysis should be strongly considered.

If a patient might need dialysis in the next several hours, it is prudent to ensure that this is possible at your institution. For example, if a small hospital has no capabilities for emergent hemodialysis, and you think that such a treatment may be warranted in the next several hours, transferring the patient to a facility capable of performing emergent hemodialysis is appropriate. Similarly, consideration should be given to placing a temporary dialysis catheter as soon as possible—this may be done in the emergent department or immediately upon admission.

In summary, the decision to perform dialysis is a complex one that incorporates both clinical and laboratory data, and circumstances may change quickly, depending on the patient's response to medical therapy. While it is important not to delay a needed therapy, emergent dialysis should be reserved for those individuals truly in need of it now.

## KEY POINTS

- Indications for emergent hemodialysis include pulmonary edema, hyperkalemia, or metabolic acidosis refractory to medical management, uremia, or removal of toxins.
- Neurotoxicity is the primary indication for dialysis in lithium toxicity.
- Indications for hemodialysis in salicylate toxicity include failure of medical management, neurologic compromise attributed to the salicylate toxicity, pulmonary edema, renal failure, and refractory acid-base disturbances.

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# **TO THRILL OR NOT TO THRILL: WHEN DIALYSIS ACCESS SITES GO WRONG**

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**CAROLINE BRANDON, MD**

Kidney disease is the ninth leading cause of death in the United States. Over 450,000 people are on dialysis, which means that these patients are not infrequently in the emergency department (ED). Many times, these patients present with complaints surrounding their vascular access for dialysis. There are five major complaints associated with arteriovenous (AV) fistulas that you must watch out for and be prepared to treat. They are thrombosis, stenosis, aneurysm, infection, and bleeding.

Primary AV fistulas are the most ideal form of vascular access; however, in certain patient populations, the outcome is less than ideal. 30% to 50% of fistulas never mature to be used for dialysis. Thus, 2/3 of patients have native fistulas—the rest have graft fistulas.

Thrombosis occurs in 9% of AV fistulas and 25% of grafts. Thrombosis can present with pain at the fistula site and a palpable thrombus. This is often preceded by stenosis. When examining the fistula in the ED setting, note whether the fistula collapses with arm elevation. A noncollapsing fistula is associated with a flow obstruction. Noting the absence of a thrill over the access site is important as well because this is a cardinal sign of outflow obstruction. The bruit and thrill should be heard and felt throughout the entire fistula. Their absence may be an indication flow obstruction from stenosis or thrombosis.

When stenosis is identified in the absence of an accompanying thrombosis, either angioplasty or pharmacologic treatments can be utilized to

prevent or delay thrombosis. Medications include aspirin, clopidogrel, dipyridamole, warfarin, and, less known agents, fish oil and angiotensin converting enzyme (ACE) inhibitors. Interventions include balloon dilatation, stent implantation, or fistula revision. Patients with thrombosis or severe stenosis require admission and/or appropriate surgical consultation in the ED.

Aneurysms commonly occur from repeated cannulation attempts, which result in a pathologic enlargement of the vessels. These can be confused with a pseudoaneurysm, which stems from a hematoma around the vessel itself but can eventually form a cavity connected to the vessel. These two entities can be differentiated with ultrasound using color Doppler. If an aneurysm is large enough or there is a high enough risk of bleeding, surgical repair is indicated. Caution should be exercised with further vascular punctures to avoid worsening the aneurysm. If the access site is bleeding briskly, remember that saving the patient is more important than the graft or fistula. A tourniquet is a good temporizing measure to definitive surgical therapy. Indications for an emergent vascular consultation for aneurysm include an increase in size >10% over a year, simultaneous infection, skin ulceration or scabbing, and anastomotic leak. Other aneurysms can be managed on an outpatient basis.

Bleeding-related fatalities account for 0.4% of all dialysis-related deaths. In a retrospective study looking at fatalities related to vascular access hemorrhage, patients who were at higher risk of bleeding were those with recent infections, previous bleeds, and evidence of graft erosion. They can be intimidating to manage, but should be controlled in the following stepwise manner. First, apply direct pressure. Remember, the fistula won't be of much use if the patient exsanguinates. If the bleeding stops with direct pressure, discharge is appropriate after a 1- to 2-hour period of observation and return precautions. If it does not, many options exist and may be helpful. These include protamine sulfate to reverse heparin, topical gel foam, thrombin, thromboxane (TXA), aminocaproic acid, and/or desmopressin (DDAVP). These patients should all be admitted for vascular surgery evaluation.

Lastly, dialysis access-related infections are the number one cause of morbidity and mortality in dialysis patients. In one study comparing the risk of death due to infection in dialysis patients compared to the general public, there was a 100- to 300-fold increase in patients on dialysis. Infections account for 20% of fistula complications and are more frequent in grafts and dialysis catheters. Infections range from superficial skin infections to infected aneurysms or hematomas to abscesses. *Staphylococcus* species are the number one culprit infections. Treatment should be initiated in the ED with vancomycin and either gentamicin or a third-generation cephalosporin

and should be continued for 3 to 4 weeks.

## KEY POINTS

- Complications of dialysis access need to be recognized promptly and dealt with appropriately.
- A physical exam should be performed on the dialysis access site.
- Complete absence of a bruit and thrill over an access site warrants admission and/or an urgent vascular consultation in the ED.
- In brisk hemorrhage, the patient's life comes before the survival of the access site.

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# **WRAP YOUR HEAD AROUND THIS: AVOIDING THE PITFALLS OF PHIMOSIS AND PARAPHIMOSIS MANAGEMENT**

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**KELLY A. PAINTER, MD, FACEP**

Delays in diagnosing and managing phimosis and paraphimosis can have devastating consequences for your patient. As time is myocardium in acute myocardial infarction, time is penis when immediate reduction of a paraphimosis is required.

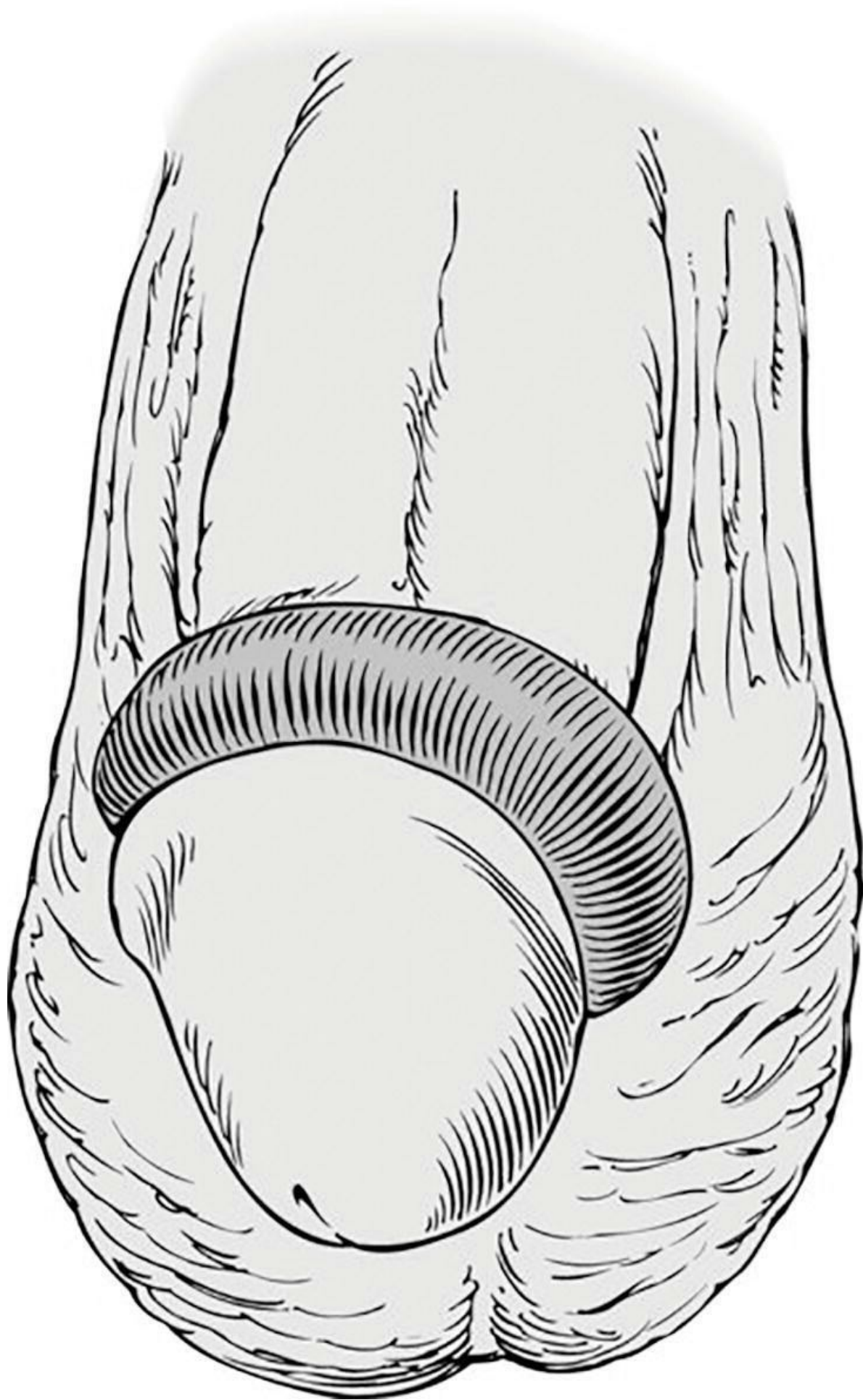
Phimosis is the inability to retract the distal foreskin over the glans penis. Most cases in newborns are physiologic and do not require emergent intervention. Physiologic phimosis will resolve spontaneously in 90% of boys by 4 years of age. If the phimosis persists beyond age 4, topical steroids with betamethasone for 6 weeks have been found to be effective and may prevent the need for circumcision.

Acquired phimosis is the inability to retract the foreskin after it was previously retractable, most often due to distal scarring of the foreskin. These acquired cases are usually caused by recurrent balanoposthitis, poor hygiene, or forcible retraction of the foreskin. The good news is that patients with acquired phimosis rarely require any emergency intervention. One pitfall to avoid, however, is discharging a patient with urinary outlet obstruction due to phimosis. To ensure this does not happen, have the patient urinate and perform a bedside bladder ultrasound to check for urinary retention. If urinary retention does occur, this may be treated with gentle dilatation of the prepuce with forceps and urethral catheter placement. All patients with



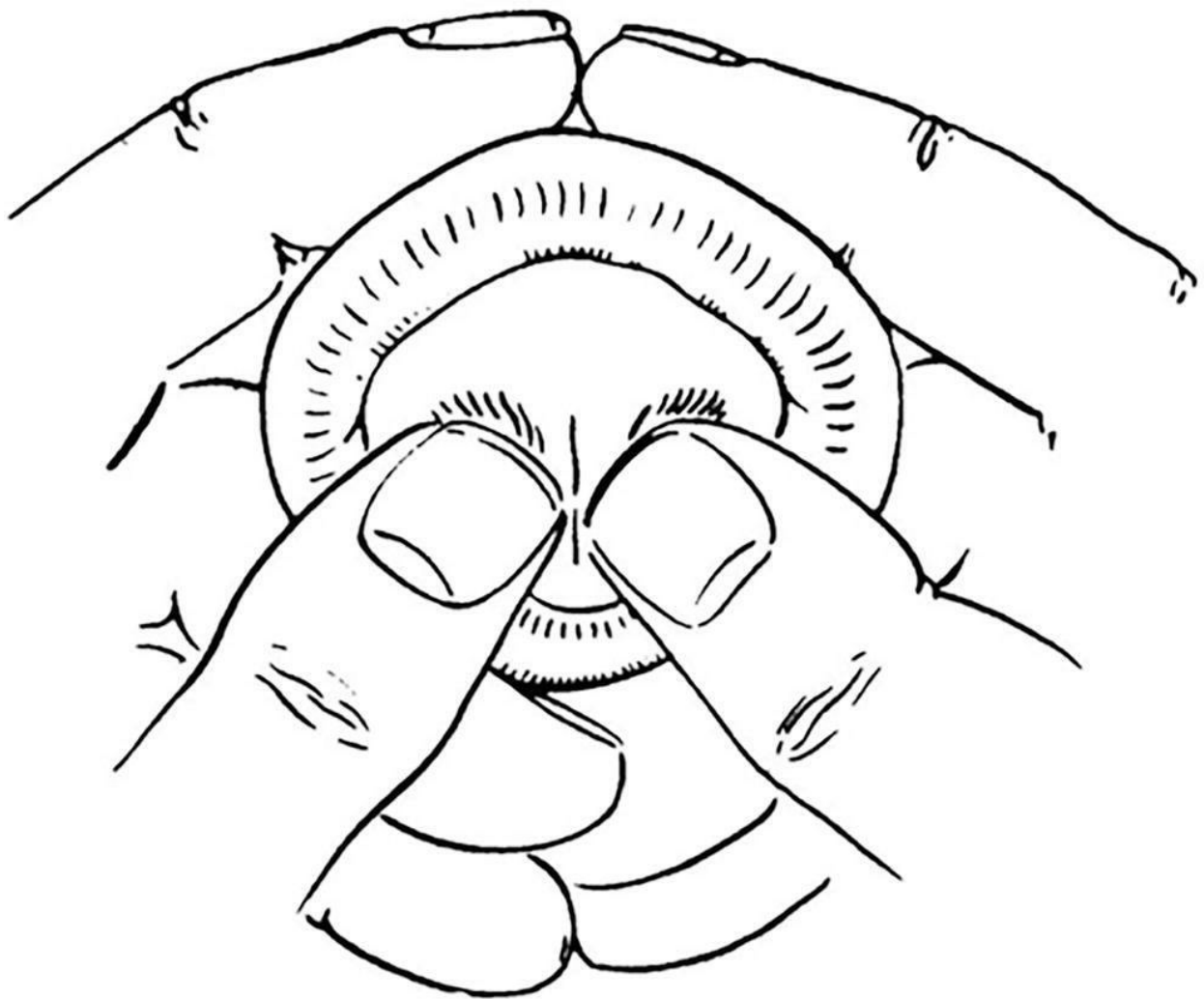
acquired phimosis should be referred to a urologist.

Paraphimosis is a rare condition in which the foreskin of an uncircumcised male becomes trapped in the retracted position proximal to the glans ([Figure 226.1](#)). The most common cause of paraphimosis is prior phimosis that results in circular scar formation at the distal end of the foreskin. This acts as a tourniquet when the foreskin is retracted. Another significant cause is iatrogenic—when a health professional fails to reduce the foreskin after urethral catheterization. Ultimately, the constricting ring of foreskin impairs venous flow causing edema to the glans. As the edema worsens, arterial blood flow is impaired, and ischemia, gangrene, or autoamputation of the distal penis may occur. Patients typically present with significant pain to the glans and inability to retract the foreskin proximally or distally. This can look just like a hair tourniquet—so don't forget to ask if the patient is actually circumcised!



**Figure 226.1** Paraphimosis. (From Weber JR, Kelley JH, eds. *Health Assessment in Nursing*. 5th ed. Philadelphia, PA: Wolters Kluwer, 2013.)

Paraphimosis is an extremely painful condition that requires parenteral analgesia and often a dorsal penile nerve block as well. Following this, immediate manual reduction should be attempted. Manual reduction is performed by placing both thumbs over the glans with both index and long fingers surrounding the trapped foreskin. The glans is then pushed back through the prepuce with constant thumb pressure while the index and long fingers pull the foreskin over the glans ([Figure 226.2](#)). If manual reduction is unsuccessful, there are multiple techniques described to decrease edema and facilitate reduction ([Table 226.1](#)). The biggest pitfall in managing paraphimosis is to forget that time is penis. Do not wait hours for your urologist to call back. The edema will worsen with time and make reduction more difficult. After successful reduction, if the patient is able to void, outpatient follow-up with urology is indicated and the patient may be discharged. If manual reduction attempts fail, emergent urologic consultation should be obtained and the patient may require a dorsal slit procedure or circumcision.



**Figure 226.2** Reducing a paraphimosis. Manual reduction of paraphimosis. After a local anesthetic block of the dorsal nerve of the penis, the foreskin is manually compressed to reduce edema. The foreskin can be reduced by pressure on glans—like turning a sock inside out.(From Klauber GT, Sant GR. Disorders of the male external genitalia. In: Kelalis PP, King LR, Belman AB, eds. *Clinical Pediatric Urology*. 2nd ed. Philadelphia, PA: WB Saunders, 1985:287. Reprinted with permission.)

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|---|
| <p><b>TABLE 226.1 TECHNIQUES FOR MANUAL REDUCTION OF PARAPHIMOSIS (LEAST INVASIVE TO MOST INVASIVE)</b></p> |
|---|

1. Ice: Soak the penis in a glove full of ice for 5 min.
2. Compression: Wrap an elastic bandage from the glans to the base of the penis for 5 to 7 min.
3. Osmotic: Soak a swab in 50 mL of 50% dextrose and wrap around glans and foreskin for 2 h.
4. Puncture: Use a 24-gauge needle to puncture tiny holes in the foreskin and manually compress to allow edema to escape puncture sites.
5. Hyaluronidase: Use a Tb syringe to inject 1 mL aliquots of hyaluronidase into the prepuce.
6. Aspiration: Apply tourniquet to shaft of the penis and use a 20-gauge needle to aspirate blood from the glans parallel to the urethra.

## KEY POINTS

- Ensure that patients are able to urinate prior to discharge.
- Paraphimosis is a urologic emergency that requires immediate intervention.

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## PYELONEPHRITIS: WHEN IT'S COMPLICATED URINE TROUBLE

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MOLLY HARTRICH, MD, MPH AND SOPHIE TERP, MD, MPH

Pyelonephritis is an infection of the upper urinary tract including the kidneys and ureter, typically resulting from an ascending lower urinary tract infection (UTI). Rarely, descending infections may result from hematogenous spread in bacteremic patients. Clinical symptoms of pyelonephritis include fever, back pain, nausea, vomiting, malaise, confusion (especially in the elderly), as well as painful, urgent, or frequent urination. All patients suspected of having pyelonephritis should have urine culture and sensitivity testing performed and empiric treatment initiated pending availability of culture results. *Escherichia coli* is the pathogen responsible for 75% to 95% of UTIs. Antibiotic resistance patterns vary considerably between regions, and treatment selection should reflect patient allergies, medication interactions, and local resistance patterns. Without prompt and appropriate treatment, pyelonephritis may be associated with significant morbidity—this is due to its natural progression to sepsis in many cases. Providers can optimize care of patients with pyelonephritis in the emergency department (ED) by avoiding the common management errors described below.

***One common error in the ED management of pyelonephritis is use of antibiotics with poor renal tissue penetration.*** Although frequently used to treat cystitis, nitrofurantoin should not be used for patients with pyelonephritis because adequate renal tissue levels are not achieved. When cystitis symptoms are accompanied by subjective fever, vague flank pain, mild costovertebral angle tenderness, or a prolonged duration of symptoms (>5 to 7 days), early pyelonephritis should be considered. In the event that cystitis cannot clearly be distinguished from early pyelonephritis,

nitrofurantoin and other medications with poor renal penetration such as fosfomycin should be avoided.

***A second common error in ED management is failure to appropriately distinguish between uncomplicated and complicated pyelonephritis.*** ED disposition often depends upon whether the infection is uncomplicated or complicated. Uncomplicated pyelonephritis occurs in young, healthy, immunocompetent, nonpregnant women without known structural or functional abnormalities of the urinary tract. Most patients with uncomplicated pyelonephritis who are able to tolerate oral medications and do not appear to be septic can be managed as outpatients with oral antibiotics and follow-up of urine culture results. Common outpatient antibiotic regimens for uncomplicated pyelonephritis include ciprofloxacin 500 mg twice daily for 7 days, levofloxacin 750 mg daily for 5 days, or trimethoprim-sulfamethoxazole 160/800 mg twice daily for 14 days. There is a trend toward shorter treatment courses in recent years—consult up-to-date guidelines when prescribing.

Any case of pyelonephritis occurring in a man or in a woman who is pregnant, diabetic, immunosuppressed (e.g., AIDS), has a genitourinary abnormality whether functional (e.g., neurogenic bladder) or structural (e.g., nephrolithiasis), or has any underlying medical condition that increases the risk of infection is classified as complicated. In these patients, the risk of treatment failure is greatly increased. While patient disposition is ultimately up to the clinical discretion of the emergency provider, ED observation or inpatient admission for IV antibiotics and close monitoring should be strongly considered for patients with complicated infection. Patients requiring hospitalization should be initially treated with an intravenous antimicrobial regimen such as a fluoroquinolone; an aminoglycoside, with or without ampicillin; an extended-spectrum cephalosporin or extended-spectrum penicillin, with or without an aminoglycoside; or a carbapenem.

Pregnant patients should be treated aggressively, as pyelonephritis may induce preterm labor and progresses to sepsis relatively frequently among pregnant women. Inpatient admission for intravenous antibiotics should be considered in most pregnant patients with pyelonephritis although outpatient therapy is increasingly accepted in well-hydrated patients with reliable access to follow-up. Cephalosporins are used most frequently for pregnant patients with pyelonephritis. Aztreonam can be used for pregnant patients with severe penicillin or cephalosporin allergies. Tetracyclines, fluoroquinolones, aminoglycosides, trimethoprim, and sulfonamides should be avoided in pregnancy.

***A third common error in ED management of pyelonephritis is failure***



***to provide adequately broad antibiotic coverage for patients with known (or at high risk for) infection with drug-resistant organisms.*** Given the increasing prevalence of UTIs due to multidrug-resistant pathogens such as extended-spectrum beta-lactamase (ESBL)-producing organisms and carbapenem-resistant *Enterobacteriaceae* (CRE) infections, when available, results of prior urine cultures and susceptibilities should be used to guide antibiotic selection. For patients with history of infection with an ESBL-producing organism, carbapenems are the drug of choice. Initiating an effective treatment regimen for a patient with history of prior CRE infection may require consultation with a pharmacist or infectious disease specialist. When prior culture results are unavailable, broad-spectrum antibiotics should be considered for those at risk for infection with multidrug-resistant organisms including frequently hospitalized patients, transplant recipients, and residents of skilled nursing facilities.

***A final common error in ED management is failure to identify complications of pyelonephritis requiring procedural intervention.*** For patients with urosepsis or shock, urgent imaging should be obtained to exclude obstructing ureteral stone or other surgical emergency such as emphysematous pyelonephritis. Bedside ultrasound can be used to evaluate for hydronephrosis before obtaining a CT to evaluate for obstructing ureterolithiasis. ED providers should consider obtaining cross-sectional imaging to evaluate for renal abscess or emphysematous pyelonephritis in ill-appearing septic patients without appropriate clinical response to antibiotics and fluid resuscitation, especially if the patient is diabetic. If any of these are identified, inpatient admission with urgent urology consultation should be arranged.

## KEY POINTS

- Avoid use of antibiotics with poor renal penetration when upper UTIs cannot be excluded.
- Accurately identify and appropriately treat cases of complicated pyelonephritis.
- Base antibiotic choices on prior culture results when available and provide appropriately broad coverage to patients at increased risk for infection with resistant organisms.
- Consider and evaluate for complications of pyelonephritis requiring procedural intervention.



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# WHAT GOES UP MUST COME DOWN

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**JESSICA LANGE OSTERMAN, MS, MD**

Priapism is a sustained erection, generally lasting >4 hours, in the absence of sexual stimulation or persisting after the cessation of sexual stimulation. It is a rare but potentially devastating condition that can result in permanent erectile dysfunction for the patient and should be considered a urologic emergency when it presents to the emergency department.

Perhaps the most common error in the management of priapism is the physician's failure to establish the etiology of priapism. There are two main types of priapism, ischemic and nonischemic, and their treatment differs significantly. Therefore, it is important for the treating physician to identify the type expediently to facilitate treatment. Ischemic, or low-flow, priapism is far more common and is a urologic emergency that must be treated emergently to avoid permanent structural damage to the penis and permanent erectile dysfunction. In ischemic priapism, there is an accumulation of venous blood in the corpora, which leads to venous congestion, pain, ischemia, and eventually fibrosis. Ischemic priapism is essentially a compartment syndrome of the penis, and if it lasts longer than 24 hours, it is associated with up to a 90% rate of subsequent erectile dysfunction. The ischemic form is most often due to medication usage but can also be related to underlying conditions such as sickle cell disease, hyperviscosity syndromes, or malignancy. Nonischemic, or high-flow, priapism is the rarer of the two forms and is caused by abnormal arterial inflow into the cavernosa. Nonischemic priapism is often associated with trauma and is typically either pain-free or significantly less painful than the ischemic form. Aspiration and blood gas analysis of corporal blood are reliable methods for determining the category of priapism and can be performed if there is any

clinical question which type is present. If ischemic, corporal blood should be hypoxic, hypercarbic, and acidotic (generally  $P_{O_2} < 30$  mm Hg,  $P_{CO_2} > 60$  mm Hg,  $pH < 7.25$ ). In the nonischemic form, the cavernous blood gas should reflect normal arterial blood. Treatment should be initiated expediently after determining the type of priapism present in the patient.

After establishing the type of priapism, another possible error is the failure to evaluate the underlying etiology for the condition when no obvious cause exists. In cases of ischemic priapism without a distinct history of contributory medications or drugs, a thorough workup should be done to rule out other associated conditions. Ischemic priapism may be the presenting symptom of a new malignancy such as lymphoma or be associated with hyperviscosity syndrome from another underlying malignancy. Likewise, in children, it can be the first presentation of sickle cell disease. In these situations, laboratory studies such as a complete blood count or hemoglobin electrophoresis may be helpful in establishing an underlying condition or cause.

Finally, the successful and emergent treatment of ischemic priapism is essential for restoring normal erectile function and avoiding significant morbidity. First-line therapy in the treatment of ischemic priapism focuses corporal aspiration of venous blood as well as the instillation of alpha-adrenergic agonists, such as phenylephrine, into the corpora. The key to successful treatment is appropriate anesthesia to facilitate corporal drainage and detumescence; don't make the error of inadequately anesthetizing the patient. Anesthesia should be attained through performance of a dorsal penile block or a local penile shaft block. The dorsal penile block targets the left and right dorsal penile nerves that run at ~2 o'clock and 10 o'clock positions at the base of the penis. After cleansing, the physician should start by anesthetizing the skin over the 2 and 10 o'clock with superficial wheals of lidocaine without epinephrine (although some controversy exists about the use of epinephrine in these penile blocks). The needle should then be inserted about 0.5 cm into the skin or until the needle enters Buck fascia at the 2 and 10 o'clock positions. After aspirating to ensure the needle is not in a vessel, ~2 mL of lidocaine should be instilled at each location. A complete block will greatly facilitate the ease of aspiration and instillation of medications to achieve detumescence and restore normal penile function.

Treatment of nonischemic priapism is completely different. Since the nonischemic form is associated with the flow of well-oxygenated blood, there is no indication for removal of blood from the corpora or instillation of vasoconstrictors. This type generally does not necessarily represent an emergency, as outcomes do not worsen over time. Nonischemic priapism

may actually improve spontaneously; thus, it may be treated with a period of observation after urgent referral to urology.

## KEY POINTS

- First identify the subtype of priapism present.
- Investigation into the underlying cause can help guide treatment and the urgency of urologic intervention.
- Adequate anesthesia with a dorsal penile block will facilitate successful aspiration and aid in the restoration of normal penile function.

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# STREAMLINING URETHRITIS: DON'T LET AN STD ESCAPE YOUR ED

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CLARE ROEPKE, MD

Urethritis is an inflammation of the lower urinary tract that can be caused by infectious and noninfectious conditions. It presents in both men and women and is a common complaint in the emergency department (ED).

The symptoms and management of urethritis in men are distinctly different from those in women. In men, the usual presenting complaint is dysuria. Other common complaints include burning, pruritis, and discharge at the urethral meatus. Discharge may be present throughout the day or only with the first morning void. Men may also note a crusting of the urethral meatus when they wake or an ability to express discharge. Most literature focuses on the infectious causes of urethritis including *Neisseria gonorrhoea* (GC), *Chlamydia trachomatis*, *Mycoplasma genitalium*, *Ureaplasma urealyticum*, *Trichomonas vaginalis*, and *M. genitalium*, with *N. gonorrhea* and *C. trachomatis* being the most common.

If a male patient is symptomatic with urethritis, your workup starts with a clean-catch urine specimen to evaluate for GC and chlamydia (DNA-based testing). This test will not result immediately; however, determining the specific etiology of the urethritis is both important and recommended in order to prevent complications, reinfection, and transmission of disease. So where do you go from here?

Firstly, the diagnosis of urethritis can be confirmed in a symptomatic male with the following findings:

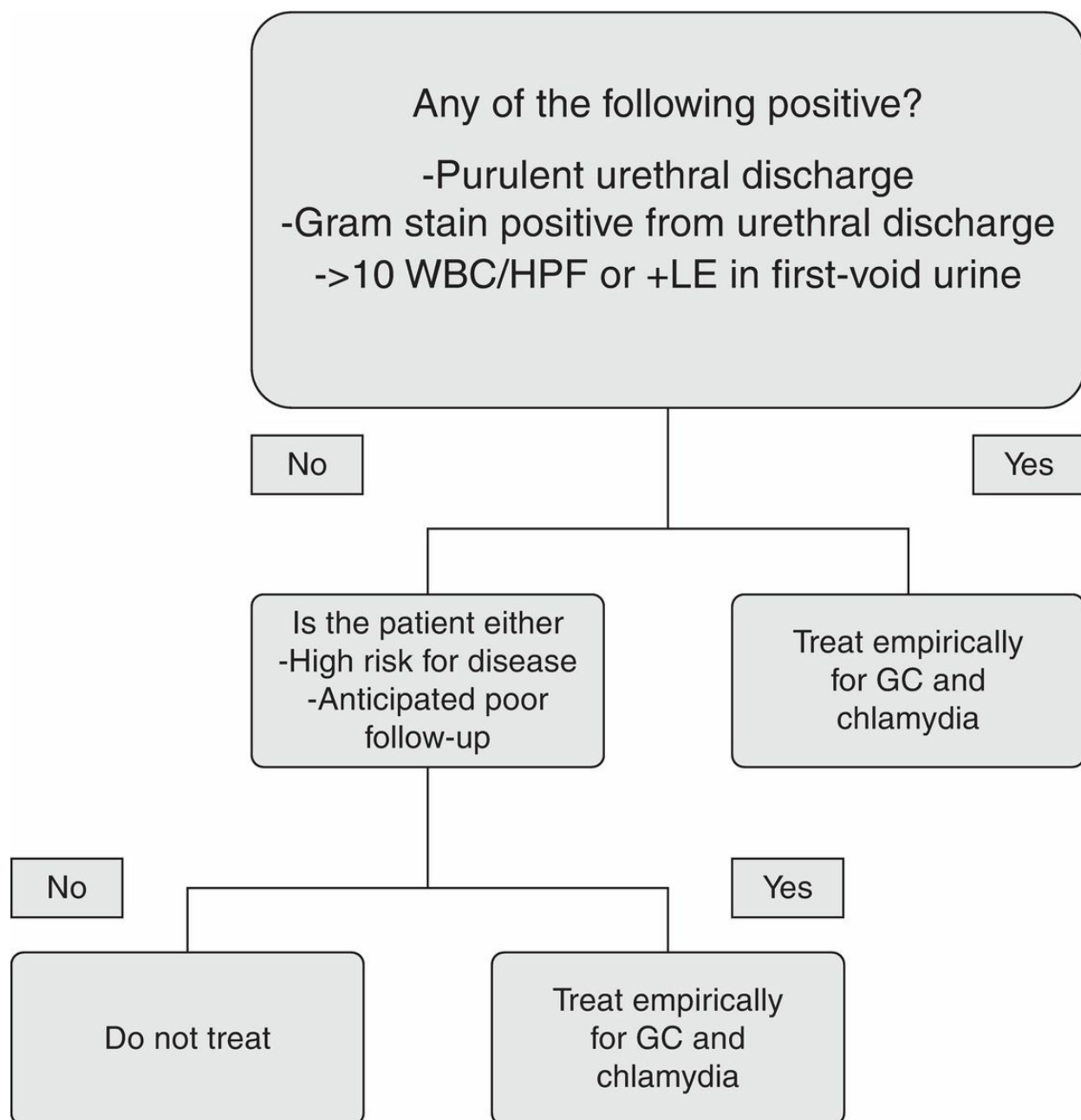
- Mucopurulent or purulent discharge on exam
- Positive leukocyte esterase test or >10 white blood cells per high-power field in sample of first-void urine
- Gram stain of urethral secretions demonstrating >2 white blood cells per oil immersion field or intracellular diplococci

Thus, if there is no discharge present on your exam and your ED does not have the capability of performing a gram stain from a urethral swab, a first-void urine specimen should be obtained to identify pyuria.

Once the diagnosis of urethritis is confirmed, remember that the DNA-based testing to detect GC and chlamydia may not yield results for more than 24 hours. So what do you do in the meantime? Should you empirically treat everyone with urethritis for sexually transmitted infections (STIs)? The following principles should guide your treatment decisions:

Men who meet at least one criterion for urethritis listed above should be empirically treated for GC and chlamydia. Men evaluated who have symptoms of urethritis but no clinical findings (e.g., no urethral discharge, first-void urine negative for leukocytes) should only be empirically treated if the man is considered high risk (defined as having unprotected intercourse, multiple partners, a high-risk partner, or a history of STIs or intravenous drug abuse). Empiric treatment should also be considered for patients unlikely to return for a follow-up evaluation or test results ([Figure 229.1](#)).

The recommended treatment regimen aimed at treating both GC and chlamydia (given the high rate of coinfection) is ceftriaxone 250 mg IM ×1 and azithromycin (1 g PO ×1) or doxycycline 100 mg PO b.i.d. ×7 d. The goals of treatment are to alleviate symptoms, prevent complications, reduce transmission of coinfections, and identify and treat close contacts. To decrease the rates of transmission and reinfection, men treated for GC and chlamydia should be instructed to abstain from sexual intercourse for 7 days (whether that means the 7 days of treatment or for 1 week after single-dose therapy), until symptom resolution and until their partner is adequately treated as well.



**Figure 229.1** Management of urethritis in men.

## KEY POINTS

- Send GC/chlamydia DNA-based testing on all men with symptoms of urethritis.
- If urethritis is confirmed by purulent discharge, positive gram stain of discharge, or abnormal first-void urine, empiric STI treatment is warranted.
- Risk factors for disease and compliance must be considered—empiric therapy may be appropriate in high-risk patients.

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# **I DON'T THINK MY URINE IS SUPPOSED TO LOOK LIKE THIS!**

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**LANDON A. JONES, MD**

Hematuria is not a sexy topic. It is an important topic, though, and one that every emergency practitioner will see many times in their career. Few complaints bring a patient to the emergency department faster than “peeing blood,” and the first step in addressing this issue involves identifying the rare patient with significant blood loss. For the remaining patients, the key is providing reassurance—it takes very little blood to turn the urine red. As little as 1 mL of blood can discolor 1 L of urine. Literature on hematuria tends to focus on glomerular (kidney-related) versus nonglomerular (urologic) causes. It is important, though, to take a step back and look at the big picture when addressing suspected hematuria. The process of hematuria evaluation can be simplified into three broad questions:

## **IS THE PATIENT ABLE TO URINATE?**

Clots are the most common cause of urinary blockage. If the patient is unable to urinate, significant electrolyte abnormalities (hyperkalemia, elevated creatinine) need to be considered. If the patient is unable to urinate and can be catheterized, a 3-lumen irrigation catheter is best. One lumen inflates the balloon that is used for securing the catheter in the bladder. The second lumen allows urine to drain and the third lumen can be used for irrigation of potential clots. Placement of a catheter also offers significant pain control in a patient that has had the inability to void.

**DOES THE PATIENT ACTUALLY HAVE**

## HEMATURIA?

Hematuria is defined as the presence of red blood cells (RBCs) in the urine. There are a number of causes, aside from hematuria, that can change the color of the urine. These include, but are not limited to, myoglobin, hemoglobin, bilirubin, foods, drugs, and dyes. For instance, a patient with rhabdomyolysis and the production of urinary myoglobin may have a positive urine dipstick and dark-colored urine but no RBCs on urine microscopy. If hematuria is confirmed, it usually is described as microscopic hematuria (>5 RBCs/HPF and only seen on urine microscopy) or macroscopic hematuria (visible with the naked eye).

## WHY—AND FROM—IS THE PATIENT HAVING HEMATURIA?

More common causes of hematuria are urinary tract infections, urolithiasis and trauma. The patient's story is the most important part of the assessment and should include questions such as duration of hematuria, circumstances (including trauma and recent instrumentation/surgery), and associated signs/symptoms. It is also important to ask about potential causes of coagulopathy such as anticoagulation use, hemophilia, thrombocytopenia, and family history of a bleeding diathesis. As mentioned above, hematuria causes are commonly broken down into glomerular (kidney-related) and nonglomerular (urologic) causes. It may be easiest for the practitioner to simply start with the kidneys and work down the urologic system to the urethra and external genitalia. Further evaluation with labs and/or imaging will be dictated by the practitioner's history and physical examination. A complete blood count can detect thrombocytopenia. Electrolytes may need to be assessed if there is concern for significant renal impairment. Coagulation studies may be warranted for patients on warfarin. Urine studies, including a pregnancy test in appropriately aged females, should be obtained. Imaging modalities will also be dictated by the history and examination. Ultrasound or computed tomography (CT) imaging may be necessary.

There are two big pitfalls when addressing hematuria. The first (and most dangerous) is to *assume* that the hematuria is from a renal or genitourinary (GU) source. While renal/GU sources are by far the most common, life-threatening vascular issues can also cause hematuria. It is worthwhile to pause and consider such rare diagnoses briefly before moving to discharge these patients. The medical literature has numerous reports of aortic compression, aneurysm, fistula, and dissection causing hematuria.

Fortunately, serious vascular causes of hematuria commonly have other physical signs such as abdominal tenderness, lower extremity neurologic deficits, and asymmetric lower extremity pulses. This underscores the importance of performing a good physical examination.

The second biggest pitfall is frankly just to ignore it. Even though many cases of hematuria spontaneously resolve, timely follow-up with repeat urinalysis must be recommended to confirm resolution. Unresolving hematuria requires further evaluation in the outpatient setting because renal/urologic malignancy is high on the list of differential diagnoses, especially in older patients. Some studies have demonstrated that macroscopic hematuria has an above-average sensitivity for urologic malignancy and about 30% of patients with painless hematuria are diagnosed with urologic malignancy. While extensive workup of hematuria is infrequently required in the emergency department setting, it is important to inform and educate the patient, making them aware that hematuria needs to be followed up in a timely fashion in an outpatient setting.

## KEY POINTS

- Recognize potential life-threatening electrolyte abnormalities early. Renal insufficiency/failure can occur in the setting of hematuria and these patients can decompensate quickly.
- Confirm the presence of hematuria on microscopic examination. Other entities can turn urine red.
- Life-threatening vascular disorders can present with hematuria. Do not just assume hematuria is from a GU source.
- Stable patients with hematuria need outpatient follow-up due to the risk of malignancy. Emphasize this to the patient.

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# SECTION XVII

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## THORACIC

# DO NOT FORGET TO ADMINISTER STERIODS IN PATIENTS WITH ACUTE ASTHMA EXACERBATIONS

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**MICHELE CALLAHAN, MD**

Asthma is a chronic inflammatory airway disorder affecting more than 22 million Americans. Acute asthma exacerbations accounted for 1.8 million emergency department (ED) visits in 2011 alone. Approximately 10% of ED patients with acute asthma exacerbations will require hospital admission, while 12% to 16% of those discharged will return to the ED within 2 weeks for a relapse or persistence of symptoms. The recommendations within this chapter are derived from a combination of existing guidelines, clinical evidence, and practitioner consensus.

The National Heart, Lung, and Blood Institute Guidelines for the Diagnosis and Management of Asthma define asthma exacerbations as “episodes of progressively worsening shortness of breath, cough, wheezing, and chest tightness, or some combination of these symptoms.” The underlying processes that drive an acute asthma exacerbation are airway inflammation, airway hyperreactivity, and bronchoconstriction. Over time, these processes lead to airway remodeling with progressive, irreversible loss of lung function.

Goals of treatment during an asthma exacerbation are to reverse airflow obstruction, correct hypoxemia, and decrease the inflammatory response. As such, the cornerstones of ED management include inhaled short-acting beta<sub>2</sub> agonists, administration of oxygen to maintain an oxygen saturation (SpO<sub>2</sub>) >92%, and the administration of systemic glucocorticoids. The critical role of inflammation in asthma makes systemic corticosteroid administration

essential to the management of an acute exacerbation.

The Expert Panel Report 3 (EPR-3) classification of asthma severity ([Table 231.1](#)) is a useful tool to categorize patients as having a mild to moderate or severe exacerbation. This serves as a useful framework in conjunction with a provider's clinical judgment. Aspects of the history of present illness that place patients at risk for higher morbidity and mortality include previous intensive care unit admission, previous intubation, low socioeconomic status, significant comorbidities (i.e., cardiovascular disease), and more than 2 hospitalizations or more than 3 ED visits within the past year.

**TABLE 231.1 EPR-3 CLASSIFICATION OF ASTHMA SEVERITY**

| QUALITY OF EXACERBATION | FEV <sub>1</sub> OR PEF    | SPEECH                                 | PHYSICAL EXAM  | SAO <sub>2</sub> (%) | PAO <sub>2</sub> (MM HG) | HR (BPM) |
|-------------------------|----------------------------|--|--|----------------------|--------------------------|----------|
| Mild to moderate        | ≥40% of predicted function | Full sentences                         | Absent pulsus paradoxus and presence of air movement/wheezing                                    | ≥90                  | ≥60                      | ≤120     |
| Severe                  | ≤40% of predicted function | Short phrases or too dyspneic to speak | Pulsus paradoxus present and minimal air movement with near-absent breath sounds or silent chest | ≤90                  | ≤60                      | ≥120     |

FEV<sub>1</sub>, forced expiratory volume in one second; PEF, peak expiratory flow.

Adapted from the National Asthma Education and Prevention Program. Expert Panel Report 3: Guidelines for the diagnosis and management of asthma. National Heart Lung and Blood Institute, 2007:375.

Past research supports the use of systemic corticosteroids as a first-line treatment in all asthmatic patients presenting to the ED, as they have been shown to speed the resolution of airflow obstruction, reduce relapse rates, and decrease the need for hospitalization. A 2001 Cochrane Review established that systemic steroid administration to children and adults presenting to the ED with moderate or severe asthma exacerbations resulted in a 25% reduction in hospital admission. In particular, they noted that

steroid administration within 1 hour of arrival to the ED had the greatest benefit in reducing the need for hospitalization. Intuitively, this makes sense based on the time to onset of corticosteroids, which is known to be ~2 to 4 hours. A 2008 meta-analysis by Rowe and colleagues showed significant differences in relapse rates within 7 to 10 days for patients who received corticosteroids compared with patients who did not receive steroids. This study showed a number needed to treat (NNT) of just 8 for prevention of hospital admission and an NNT of 10 to decrease the relapse in the 7 to 10 days postdischarge. Additionally, the NNT to prevent one relapse to hospitalization after outpatient care was 11. The use of corticosteroids will decrease not only relapse rates but also subsequent hospital admissions in acute asthma exacerbations.

There has been little consensus about the best mode of administration (oral, intramuscular, intravenous, or inhaled), the optimal schedule (taper vs. fixed dose), or the duration of treatment with systemic steroids. No additional benefit exists when systemic steroids are given at higher doses (i.e., 60 to 80 mg or 2 mg/kg/d) compared with standard dosing. The most recent guidelines from the EPR-3 recommend 40 to 80 mg/d of prednisone (or its equivalent), divided in 1 to 2 doses. Common pediatric weight-based regimens include prednisone or prednisolone 1 to 2 mg/kg orally for 5 days (maximum dose 60 mg/d) or dexamethasone 0.6 mg/kg orally, intravenously, or intramuscularly once daily for 1 to 2 days (maximum 16 mg/dose). If intravenous dosing is necessary, methylprednisolone at a dose of 1 to 2 mg/kg may be used.

There is no proven benefit to oral steroid administration compared to intravenous or intramuscular; however, adverse reactions such as injection site pain and bruising make the intramuscular route a less favorable option. Research has shown no significant difference in relapse rates between intramuscular and oral steroid administration, particularly within the first 7 to 10 days postdischarge from the ED. Oral steroids are recommended as first-line agents, unless the patient has a decreased ability to absorb medications enterally. There are many formulations of systemic corticosteroid available based on patient factors such as these, ultimately all with similar proven efficacy.

Tapered regimens of oral steroids, in which the dose is gradually decreased over a period of several days, were previously thought to have theoretical advantage of reduced risk of adrenal insufficiency or relapse. In reality, the use of a tapered-dose regimen has equal efficacy when compared with fixed-dose schedules. Studies have failed to show differences in lung function, relapse rates, or the occurrence of adrenal insufficiency when comparing tapered-dose and fixed-dose regimens. In practice, fixed-dose

regimens of <10 days duration are often the most feasible for patients. By making dosing simpler for patients, nonadherence rates may be decreased and ultimately lower treatment failure and relapse.

## KEY POINTS

- Administration of systemic corticosteroids in the ED for acute asthma exacerbations reduces relapse rates and decreases hospitalization.
- Rapid administration of inhaled beta agonists is the primary treatment of acute exacerbations.
- High-dose steroid administration provides no added benefit over standard dosing.
- Different routes of administration have equal efficacy, with oral administration the preferred mode in patients with normal gastrointestinal absorption.
- Tapered-dose and fixed-dose regimens are equally effective.

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# DO NOT WITHHOLD OXYGEN IN A HYPOXIC PATIENT WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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**BRIAN J. LIN, MD AND ANAND K. SWAMINATHAN, MD, MPH**

It has been traditionally taught that the administration of oxygen (O<sub>2</sub>) to patients with chronic obstructive pulmonary disease (COPD) can worsen hypercarbia and lead to respiratory failure. As a result, many clinicians are hesitant to administer O<sub>2</sub> to COPD patients because of this concern. However, this classic teaching is indeed *false*.

Davies and Mackinnon first published the concept of the “hypoxic drive” in 1949, when they examined O<sub>2</sub>-induced neurologic symptoms in patients with cyanosis secondary to severe emphysema. By showing a positive correlation between cerebrospinal fluid (CSF) pressure and O<sub>2</sub> therapy, the two authors hypothesized that O<sub>2</sub> therapy led to an accumulation of carbon dioxide (CO<sub>2</sub>) and an indirect increase in CSF pressure. Furthermore, Davies and Mackinnon observed that neurologic symptoms resolved when the O<sub>2</sub> stimulus was removed. This led them to theorize that respiratory activity was dependent on anoxic stimuli and that correction of anoxia with O<sub>2</sub> resulted in hypoventilation and worsened hypercarbia.

Multiple studies in the 1980s demonstrated that O<sub>2</sub> therapy in patients with an acute COPD exacerbation caused a transient decrease in minute

ventilation and an increase in arterial carbon dioxide concentration ( $\text{PaCO}_2$ ). However, statistical analysis did not show a significant correlation between these two factors. Another study used inspiratory mouth occlusion pressure as a marker for respiratory drive and showed a higher than normal respiratory drive in COPD patients regardless of  $\text{O}_2$  therapy. These findings led investigators to conclude that while  $\text{PaCO}_2$  increased with  $\text{O}_2$  therapy in patients with acute COPD exacerbations, the increase was *not* explained by a decrease in minute ventilation or loss of respiratory drive.

Recall that in a normal physiologic state, alveolar ventilation ( $V_a$ ) and perfusion ( $Q$ ) are generally well matched. The body has compensatory mechanisms to optimize the  $V_a/Q$  ratio. When alveolar oxygen ( $\text{P}_A\text{O}_2$ ) levels decrease, local mediators promote vasoconstriction to hypoxic alveoli. This results in an increase in blood flow to alveoli with normal oxygen levels. This phenomenon is termed *hypoxic pulmonary vasoconstriction*. When high levels of  $\text{O}_2$  are administered to patients with hypoxic pulmonary vasoconstriction, the regional low  $\text{O}_2$  state is altered and hypoxic vasoconstriction is terminated. This results in restoration of perfusion to areas of poor ventilation, increased pulmonary shunt, and worsened hypercarbia. Recent research has supported the idea of  $V_a/Q$  mismatch as an explanation for  $\text{O}_2$ -induced hypercarbia in acute COPD exacerbations.

The Haldane effect is a second cause of hypercarbia after  $\text{O}_2$  administration in acute COPD exacerbations. The Haldane effect describes a property of hemoglobin and states that deoxygenated blood can carry increased amounts of  $\text{CO}_2$ . In contrast, oxygenated blood has a decreased affinity for  $\text{CO}_2$ . Thus, an increase in  $\text{P}_A\text{O}_2$  causes a decrease in  $\text{CO}_2$  bound hemoglobin and an increase in  $\text{PaCO}_2$ . This rightward shift in the  $\text{CO}_2$  dissociation curve is usually compensated for by an increase in minute ventilation to normalize the increased  $\text{PaCO}_2$ . However, since minute ventilation is generally already elevated in COPD patients, this can lead to further  $\text{CO}_2$  retention. Aubier and colleagues demonstrated that the Haldane effect can explain up to 25% of the  $\text{PaCO}_2$  increase after  $\text{O}_2$  administration.

Is there a way to avoid respiratory failure in COPD patients without withholding  $\text{O}_2$  therapy? A randomized controlled trial by Austin and colleagues compared a titrated  $\text{O}_2$  model to a high-flow  $\text{O}_2$  model. While previous retrospective studies demonstrated that high-flow  $\text{O}_2$  during acute COPD exacerbations was associated with increased mortality, Austin and

colleagues demonstrated that titrated O<sub>2</sub> therapy had a statistically significant improvement on morbidity and mortality. By aiming for an oxygen saturation of 88% to 92%, patients had less associated acidosis, requirement for assisted ventilation, and overall decreased mortality.

While O<sub>2</sub> administration can worsen hypercarbia in patients with acute COPD exacerbations, titrated O<sub>2</sub> therapy that alleviates hypoxia while avoiding hyperoxia is an appropriate treatment strategy and has been shown to decrease both morbidity and mortality.

## KEY POINTS

- Oxygen therapy should not be withheld in severely hypoxic patients with an acute COPD exacerbation.
- O<sub>2</sub>-induced hypercarbia in patients with an acute COPD exacerbation can be explained by V<sub>a</sub>/Q mismatch and the Haldane effect.
- A decrease in respiratory drive does not account for hypercarbia in the setting of O<sub>2</sub> administration in patients with an acute COPD exacerbation.
- Administer oxygen therapy to maintain an oxygen saturation of 88% to 92% in patients with acute COPD exacerbation.
- There is no difference between oxygen delivery methods when targeting goal oxygen saturation levels.

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# KNOW ACUTE ILLNESSES THAT LEAD TO RAPID DETERIORATION IN THE PATIENT WITH PULMONARY HYPERTENSION

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MD**

Pulmonary hypertension (PH) can be caused by a myriad of vascular, pulmonary, cardiac, and rheumatologic conditions. PH is defined as a mean pulmonary artery pressure (PAP) greater than or equal to 25 mm Hg at rest measured by right heart catheterization (RHC). Although echocardiography can be helpful for initial evaluation of PH, the gold standard for diagnosis remains RHC. The World Health Organization (WHO) divides PH into five groups based on the etiology ([Table 233.1](#)). The primary pathophysiologic event in PH is pulmonary vascular proliferation that results in a progressive increase in pulmonary vascular resistance (PVR). The right ventricle (RV) is a thin-walled structure with limited ability to adapt to the increase in PVR. Eventually, the RV becomes dilated with decreased contractility. In addition, RV dilatation displaces the interventricular septum into the left ventricle (LV) and impedes cardiac output. The end result is progressive RV failure. The mainstay of chronic treatment for PH remains supplemental oxygen, diuretic medications, and treatment of the underlying cause. WHO Group 1 PH is a rare disease with a unique pathophysiology and is often treated with pulmonary vasodilators ([Table 233.2](#)). Vasodilator therapy has not been found to be beneficial in other WHO Groups.

**TABLE 233.1 WORLD HEALTH ORGANIZATION CLASSIFICATION OF PULMONARY HYPERTENSION**

| WHO CLASSIFICATION OF PH                     | ETIOLOGIES   |
|--|--|
| 1. Pulmonary arterial hypertension           | CTD, portal hypertension, CHD, drug/toxin induced, idiopathic, schistosomiasis           |
| 2. PH due to left heart disease              | LV dysfunction, valvular disease   |
| 3. PH due to lung diseases and/or hypoxia    | COPD, ILD, sleep-disordered breathing  |
| 4. Chronic thromboembolic PH                 | Thromboembolic disease   |
| 5. PH with unclear multifactorial mechanisms | Hematologic disorders, sarcoid, pulmonary histiocytosis, metabolic disorders, vasculitis |

WHO, World Health Organization; PH, pulmonary hypertension; LV, left ventricular; COPD, chronic obstructive pulmonary disease; ILD, interstitial lung disease; CTD, connective tissue diseases; CHD, congenital heart disease.

**TABLE 233.2 VASODILATORS COMMONLY USED FOR WHO GROUP 1 PULMONARY HYPERTENSION**

| DRUG   | ROUTE           | ADVERSE EFFECTS  | COMMENTS   |
|--|-----------------|--|--|
| PDE-5 inhibitors<br>(sildenafil, tadalafil)  | PO              | Headache, flushing, diarrhea, vision changes, hearing loss | Used in patients with mild to severe symptoms                                |
| Endothelin receptor antagonists<br>(bosentan, ambrisentan)                                   | PO              | Hepatotoxicity, peripheral edema, headache                 | Used in patients with mild to severe symptoms                                |
| <b>Prostacyclin analogues</b> —reserved for severe symptoms and rapid progression of disease |                 |  |  |
| Epoprostenol   | IV              | Flushing, headache, jaw pain, nausea, diarrhea             | Extremely short half-life of minutes. Rebound PH with abrupt discontinuation |
| Treprostinil   | IV, SC, inhaled | IV/SC—same as epoprostenol<br>Inhaled—cough                | Short half-life of hours. Rebound PH with abrupt discontinuation             |
| Iloprost   | Inhaled         | Cough, altered taste, flushing, diarrhea, jaw pain         | Used in moderate to severe symptoms  |

WHO, World Health Organization; PH, pulmonary hypertension; PO, by mouth; IV, intravenous; SC, subcutaneous.

Patients with PH often present to the emergency department (ED) with acute RV failure. Clinically, decompensated disease manifests with signs and

symptoms that include dyspnea, hypoxia, syncope, chest pain, and lower extremity edema. Sudden deterioration in RV function can rapidly lead to shock, cardiovascular collapse, and death. In the ED, it is important to rapidly recognize and treat common precipitants of acute decompensation. These include sepsis, tachyarrhythmias, hypoxia, pulmonary embolism (PE), and the abrupt withdrawal of vasodilator medications.

Sepsis is a common cause of acute decompensation in PH patients that requires prompt recognition and treatment. It often results from bacterial translocation from the gut due to bowel edema and ischemia from chronic RV dysfunction. Central venous catheters used to administer intravenous vasodilators are another source of infection. Although systemic vasodilatation occurs in sepsis, the pulmonary circulation tends to have an *increase* in vascular tone, which can worsen RV function. Sepsis-induced hypovolemia and acidosis can also worsen RV function.

Supraventricular tachyarrhythmias (i.e., atrial fibrillation, atrial flutter) are commonly seen in patients with PH and are poorly tolerated. Atrial contraction is important to both RV and LV diastolic filling in severe PH. Maintenance of sinus rhythm, rather than simply rate control, is of utmost importance. Agents used to treat tachyarrhythmias have negative inotropic properties (i.e., calcium channel, beta-blockers) and may worsen RV function. As a result, agents such as amiodarone and digoxin are often used for atrial fibrillation. Electrical cardioversion may be required to restore sinus rhythm in many patients.

Patients with PH are at high risk of PE. Even a small PE can cause a precipitous increase in the PAP and trigger RV failure in patients with severe PH. Emergency providers should maintain a high suspicion of PE in this population. Immediate treatment with thrombolytic medications or embolectomy may be required to reverse the sudden increase in PAP and avoid cardiovascular collapse.

Hypoxia is a potent trigger of pulmonary vasoconstriction. In patients with PH, hypoxic vasoconstriction can acutely elevate the PAP and result in RV failure. Rapid and aggressive correction of hypoxemia with supplemental oxygen is important in patients with severe PH.

The abrupt discontinuation of vasodilator medications can result in a rebound increase in the PAP and precipitate RV failure. Epoprostenol has a half-life of minutes and is typically administered via a continuous infusion. Central line or pump malfunction can result in abrupt discontinuation of these medications. Such an event should be treated as a medical emergency and the infusion restarted promptly via alternate means if necessary.

## KEY POINTS

- Sepsis is a common cause of acute RV deterioration in patients with PH.
- PH patients do not tolerate atrial arrhythmias well. Electrical cardioversion may be needed to restore sinus rhythm.
- Administer supplemental oxygen to hypoxic patients with PH.
- Immediately restart continuous vasodilator infusions, most notably epoprostenol.
- Even small PEs can result in significant deterioration in the patient with severe PH.

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# **KNOW THE CRITICAL ISSUES IN RESUSCITATION OF THE DECOMPENSATED PATIENT WITH PULMONARY HYPERTENSION**

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Patients with pulmonary hypertension (PH) and right ventricle (RV) failure are prone to rapid deterioration from any insult that impairs RV function or abruptly elevates the pulmonary artery pressure (PAP). The management of critical illness in patients with PH can be challenging, and the effects of any treatment intervention on the PAP and RV must be considered.

Hypoxia induces pulmonary vasoconstriction, acutely increases PAP, and can result in rapid deterioration of the patient with PH. Supplemental oxygen should be administered to correct hypoxia. In some patients, intubation and mechanical ventilation may be required. Importantly, the patient with PH is at high risk of complications and cardiovascular collapse with airway management and initiation of mechanical ventilation. This can result from the effects of medications used for sedation and paralysis, reduced preload from positive pressure ventilation, or worsened hypoxia. In preparation for intubation and mechanical ventilation, patients should be adequately preoxygenated. Induction agents that have minimal hemodynamic effect (i.e., etomidate, ketamine) should be selected and given at a reduced dose to minimize the risk of hypotension. In addition, a neuromuscular agent with rapid onset of action (i.e., succinylcholine, rocuronium) should also be used. A vasopressor infusion should be prepared in the event that

hypotension develops during the peri-intubation period. Once the patient is intubated, acidosis and hypoxia should be aggressively corrected, as these conditions can worsen PAP and RV function. High tidal volumes and high levels of positive end expiratory pressure (PEEP) can elevate intrathoracic pressure and further worsen RV function. Hypoxia should be treated with relatively high concentrations of oxygen rather than high levels of PEEP. A goal tidal volume of 6 to 8 mL/kg of ideal body weight and a plateau pressure less than or equal to 30 cm H<sub>2</sub>O are appropriate for most patients.

Hypotension in patients with PH and acute RV failure should be aggressively treated. Hypotension can be due to hypovolemia, vasodilatation (i.e., sepsis), reduced RV contractility, or increased RV afterload. For many critically ill patients, fluid administration is often the first step in resuscitation. In the patient with severe PH, excessive fluid administration can worsen RV function and impair cardiac output. Intravenous fluids should be cautiously administered. Accurate determination of volume status in patients with PH is challenging. Central venous pressure is a poor predictor of fluid status, especially in patients with PH. Pulse pressure variation may also not be an accurate predictor of volume status or fluid responsiveness in patients with PH or RV dysfunction. Bedside echocardiography may help identify signs of RV overload, such as RV dilatation and a dilated inferior vena cava without respiratory variation. Patients with these echocardiographic findings should not receive fluid resuscitation and, in fact, diuresis may improve RV function.

Patients with RV failure often require support with inotrope and vasopressor medications. Milrinone or dobutamine can be used to provide inotropic support, but both can cause hypotension and often require the addition of a vasopressor medication (i.e., norepinephrine). Milrinone causes less tachyarrhythmias and more pulmonary vasodilatation than dobutamine and is the first-line inotropic agent for PH and RV failure. Patients with vasodilatory shock (i.e., sepsis) often require vasopressors after fluid resuscitation. All vasopressors can increase pulmonary vascular resistance and may worsen RV function. Norepinephrine has modest inotropic effects from its beta-1 adrenergic activity and may cause less pulmonary vasoconstriction than other vasopressor agents. As a result, norepinephrine is used as a first-line vasopressor in patients with PH. Phenylephrine is a pure alpha-adrenergic agonist, causes pulmonary vasoconstriction without any inotropic effect, and is typically avoided in patients with PH. Vasopressin may *reduce* pulmonary vascular tone and can be used with norepinephrine for patients with vasodilatory shock. Dopamine and epinephrine provide inotropic support and vasopressor support but are second-line agents due to the increased risk of tachyarrhythmias.

The RV is sensitive to increased afterload, and a reduction in PAP can improve RV failure. Pulmonary vasodilator therapy can be lifesaving in PH, but can also produce serious adverse effects when used inappropriately. For example, patients with forms of PH other than World Health Organization (WHO) group 1 may clinically worsen with pulmonary vasodilators. In WHO group 1 PH with RV failure, intravenous prostanoids (i.e., epoprostenol, treprostinil) are used as first-line pulmonary vasodilators. These agents are typically started in the intensive care unit with the use of a pulmonary artery catheter to monitor changes in hemodynamics in response to a slow increase in the dose of medication over days. Intravenous prostanoids can cause systemic hypotension, and inhaled pulmonary vasodilators are sometimes administered to minimize systemic vasodilatation in hemodynamically unstable patients. Inhaled nitric oxide will cause pulmonary vasodilatation and reduced RV afterload, thus improving RV function. For undifferentiated patients with PH and acute RV failure, milrinone is the most appropriate first-line pulmonary vasodilator due to its balanced hemodynamic effects. Abrupt discontinuation of intravenous pulmonary vasodilators can provoke dangerous rebound PH and should be restarted as soon as possible. In the setting of hypotension, these medications may require a careful reduction in dose. This is best done in consultation with the patient's PH specialist.

## KEY POINTS

- Use low tidal volumes and low levels of PEEP in the intubated patient with PH.
- Excessive intravenous fluid administration can worsen RV function and impair cardiac output.
- Milrinone is considered the first-line inotropic agent for patients with PH and RV failure.
- Norepinephrine is the first-line vasopressor agent for patients with PH.
- Pulmonary vasodilators infusions may need to be reduced in the setting of hypotension.

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# KNOW THE EVALUATION AND MANAGEMENT OF THE PATIENT WITH SARCOIDOSIS

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Sarcoidosis has a worldwide prevalence of 10 to 20 per 100,000. The etiology and pathogenesis of this disease remain unknown. African Americans have a 3% higher lifetime risk and present an average of 10 years earlier with symptoms, and symptoms are more acute compared to Caucasians. Ultimately, sarcoidosis is a multisystem granulomatous disorder that is characterized by noncaseating granulomas. Pulmonary involvement is the most common manifestation, but extrapulmonary manifestations are seen in up to 30% of patients. Extrapulmonary manifestations of sarcoidosis are crucial to define the extent of disease and often provide safer biopsy options during the diagnostic workup. Extrapulmonary symptoms of sarcoidosis are listed in [Table 235.1](#). Clinical suspicion should be raised if one or more of the following are present: bilateral hilar adenopathy, pulmonary reticular opacities, or skin or eye lesions.

**TABLE 235.1 EXTRAPULMONARY MANIFESTATIONS OF SARCOIDOSIS**

| <b>SITE</b>                    | <b>KEY FEATURES</b>   |
|--------------------------------|---|
| <b>Skin</b>                    | <ul style="list-style-type: none"> <li>■ In ~25%, early finding, female predominance, frequent biopsy site</li> <li>■ Many forms—papular (usually around tattoo or scar sites), nodular, or plaque-like (chronic sarcoid)</li> <li>■ More in Caucasian populations and better prognosis with high self-remission rates</li> </ul>   |
| <b>Ocular</b>                  | <ul style="list-style-type: none"> <li>■ Can cause anterior and posterior uveitis.</li> <li>■ Symptoms—dry eyes, red eyes, blurry vision, photophobia</li> </ul>  |
| <b>Upper respiratory tract</b> | <ul style="list-style-type: none"> <li>■ Can involve the pharynx, nares, and sinuses with predominant supraglottic preference</li> <li>■ Amenable to systemic or lesional glucocorticoid therapy</li> <li>■ Should be suspected with upper respiratory tract symptoms and known systemic sarcoidosis</li> </ul>   |
| <b>Cardiac</b>                 | <ul style="list-style-type: none"> <li>■ Presents as heart failure, arrhythmias, or pericardial disease</li> <li>■ Asymptomatic in up to 70% of pts</li> <li>■ Normal EKG in asymptomatic pt has high negative predictive value</li> <li>■ Definitive diagnosis with cardiac MRI</li> <li>■ Can cause sudden cardiac death and is an indication for transplant</li> </ul> |
| <b>Musculoskeletal</b>         | <ul style="list-style-type: none"> <li>■ Usually symmetric; commonly affects ankles</li> <li>■ More of a periarthrititis (swelling in soft tissue around joints) than true arthritis</li> </ul>   |
| <b>Exocrine</b>                | <ul style="list-style-type: none"> <li>■ Painless swelling of parotid and salivary glands</li> <li>■ Can occur concomitantly with Sjogren disease</li> </ul>  |
| <b>Endocrine</b>               | <ul style="list-style-type: none"> <li>■ Rare but catastrophic</li> <li>■ Frequent hypothalamic (basilar granulomatous infiltration causing meningitis) and infiltration of the thyroid</li> </ul>  |
| <b>Renal</b>                   | <ul style="list-style-type: none"> <li>■ Elevated creatinine and a bland urinary sediment in a pt with suspected or known sarcoid = sarcoid interstitial nephritis</li> <li>■ Frequent cause of nephrolithiasis—matters due to inpatient management being predominated by immunosuppression</li> </ul>  |

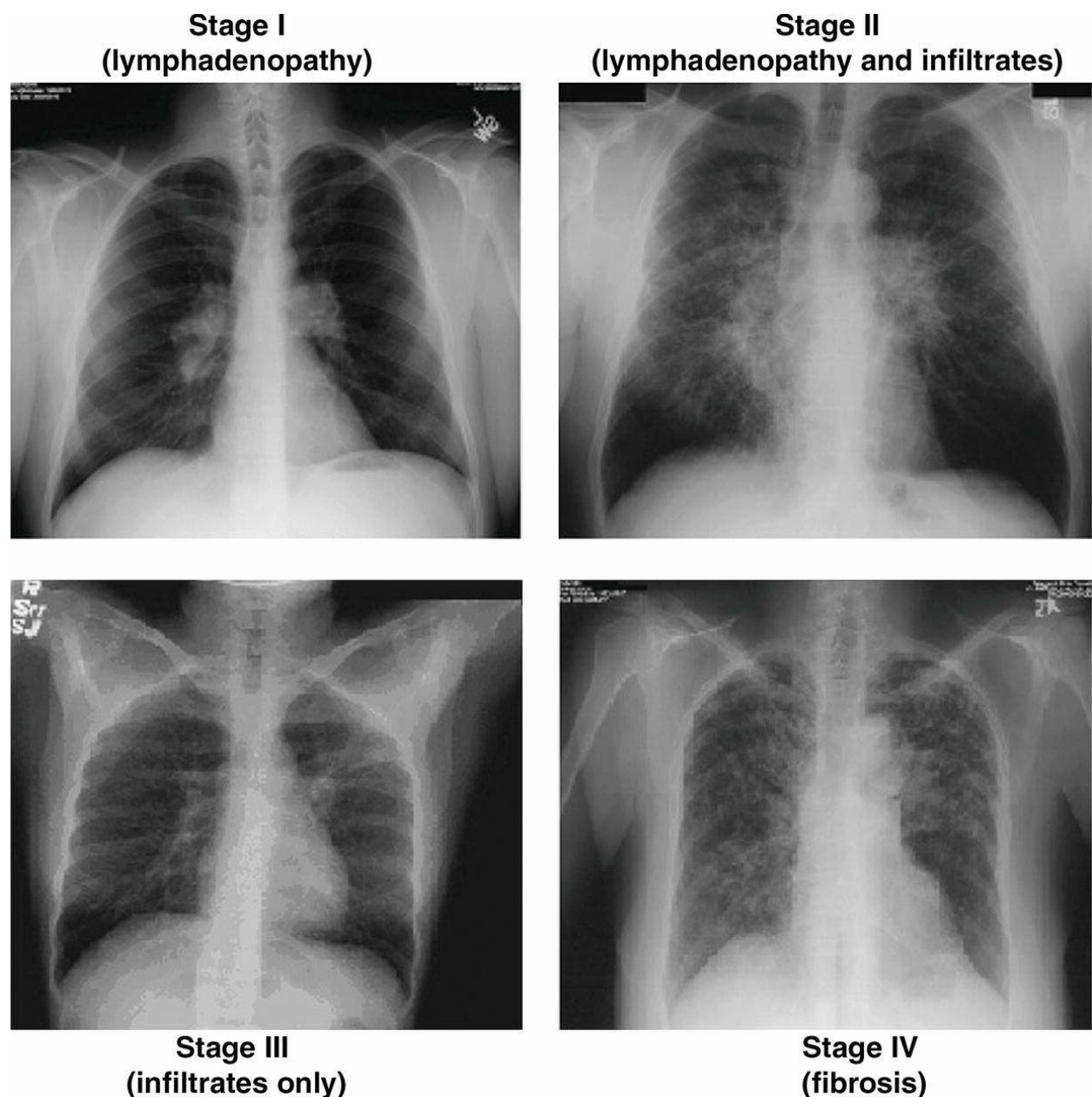
Diffuse interstitial lung disease is the most common pulmonary presentation. Symptom onset is generally between the ages of 20 and 60 years and includes cough, chest pain, and dyspnea. Older patients may present with more subtle symptoms of dyspnea but have a greater proportion of fatigue and weight loss. Radiographic abnormalities often are detected before clinical and physical exam findings. The most common radiographic progression of disease is initial hilar adenopathy with possible subsequent regression, upper lung reticular opacities, volume loss, and eventual traction bronchiectasis. High-resolution chest computed tomography (HRCT) is the next step in the evaluation of a patient with marked dyspnea, cough, and a suspicious radiograph for sarcoidosis. HRCT helps to determine the extent of parenchymal disease and to exclude other crucial diagnoses.

The next step in the evaluation of patients with suspected sarcoidosis is

referral to pulmonary medicine for outpatient pulmonary function tests (PFTs) and a 6-minute walk test. PFTs reveal a restrictive lung pattern and decreased vital capacity, total lung capacity, and diffusion of carbon dioxide. Ultimately, biopsy of the pulmonary lesions is needed to confirm the diagnosis. One major exception is Lofgren syndrome. This presents with fever, arthralgias, erythema nodosum, and bilateral hilar lymphadenopathy. The presence of all features of Lofgren syndrome has been shown to have a 95% specificity for sarcoidosis, as biopsies in the setting of this syndrome have a high false-negative rate.

Therapy is based on the extent of pulmonary and extrapulmonary involvement. While glucocorticoid therapy is the primary therapy for pulmonary sarcoidosis, adjunctive therapy with nonglucocorticoids is also indicated in certain cases. The main therapeutic considerations for the emergency provider are when to initiate steroid therapy with steroids and at what dose and duration of therapy.

Most patients with pulmonary sarcoidosis do not require therapy in the emergency department, as many are asymptomatic and have a high rate of spontaneous remission. Steroid therapy is not indicated for stage I disease (*Figure 235.1*) and for most patients with stable stage II or III disease ([Figure 235.1](#)) due to the side effects of steroid therapy. Initiation of steroid therapy is indicated for patients with debilitating symptoms and worsening radiographic finding combined with declining pulmonary function. The intent of steroid therapy is to prevent irreversible pulmonary fibrosis. High-dose steroid therapy (80 to 100 mg daily) may be indicated for cardiac, neurologic, ocular, or upper airway disease. Emergency providers should also consider adrenal insufficiency as an etiology of symptoms in sarcoidosis patients with steroid medication noncompliance or temporary inability to tolerate medications. In general, short-term steroid therapy is not sufficient, as most patients should be initiated on 0.3 to 0.6 mg/kg/d (ideal body weight) for 6 weeks. Prompt consultation with the patient's pulmonologist should be considered to coordinate care and follow-up evaluation.



**Figure 235.1** Radiograph appearance of stages I through IV.

Other immunosuppressive agents are considered in the patient who is unable to tolerate steroid side effects, failed to wean from long-term steroids, and failed to respond despite an adequate dose of steroids. The most commonly used agents include methotrexate, leflunomide, tumor necrosis factor- $\alpha$  antagonists (Remicade), azathioprine, and antimalarial agents. The initiation of such adjunctive therapy should not occur in the emergency department.

## KEY POINTS



- Consider sarcoidosis in the patient with cough, dyspnea, and hilar lymphadenopathy.
- Extrapulmonary manifestations are seen in up to 30% of patients with sarcoidosis.
- Consider other etiologies of hilar lymphadenopathy such as human immunodeficiency virus infection, occupational or environmental exposure, mycobacterium infections, fungal infections, pneumoconiosis, and hypersensitivity pneumonitis.
- Noncaseating granulomas are the hallmark of sarcoidosis.
- Initiate appropriate steroid therapy for debilitating symptoms, worsened radiographic opacities with increased pulmonary function impairment. Start with 0.3 to 0.6 mg/kg/d (ideal body weight) of prednisone.

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# PROPERLY RISK STRATIFY THE PATIENT WITH SUSPECTED PULMONARY EMBOLISM

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Venous thromboembolism (VTE) is a common cardiovascular disease with an annual incidence of 100 to 200 per 100,000. Pulmonary embolism (PE) is the most serious VTE, as it interferes with both circulation and gas exchange and can lead to death from right ventricular failure. The diagnosis of PE is challenging, as presenting signs and symptoms are nonspecific and can include dyspnea, chest pain, or syncope. Commonly obtained studies used in the evaluation of these complaints (i.e., labs, electrocardiogram, chest x-ray) do not confirm the diagnosis of PE. Furthermore, 30% of patients with a diagnosed PE have no determinable risk factor and 40% have normal oxygen saturation readings.

In order to risk stratify patients with suspected PE, it is essential to determine a clinical pretest probability. The PIOPED investigators confirmed the accuracy of a provider's "gestalt," or global clinical judgment. However, as one must have adequate experience to form an appropriate clinical judgment, there are also several clinical decision instruments that can be applied. The Wells score evaluates the presence or absence of specific clinical factors to determine the likelihood that a PE exists: signs and symptoms of DVT (3 points), an alternative diagnosis that is less likely than PE (3 points), heart rate >100 beats/min (1.5 points), immobilization or surgery in the previous 4 weeks (1.5 points), previous DVT/PE (1.5 points), hemoptysis (1 point), and malignancy (1 point). Application of a provider's clinical judgment in conjunction with an established prediction rule allows one to classify patients with suspected PE into categories of probability.

In patients with a low pretest probability of PE (Wells score < 2), it is appropriate to apply the pulmonary embolism rule-out criteria (PERC) rule. Developed by Kline and colleagues, the PERC rule advocates that PE can be excluded if all of the following eight criteria are present: age < 50 years, pulse < 100 beats/min, oxygen saturation readings >95%, no hemoptysis, no estrogen use, no surgery or trauma requiring hospitalization within 4 weeks, no prior VTE, and no unilateral leg swelling. There are three common pitfalls that must be avoided when applying the PERC rule. First, a patient must meet all eight criteria or the sensitivity of its application decreases. In addition, this rule is only valid in patients with a low pretest probability of PE and should not be applied in the moderate and high probability groups. Finally, there were patients excluded from the initial PERC trial, including cancer patients and those with personal or a family history of thrombophilia, so its application may be unreliable in these patient populations.

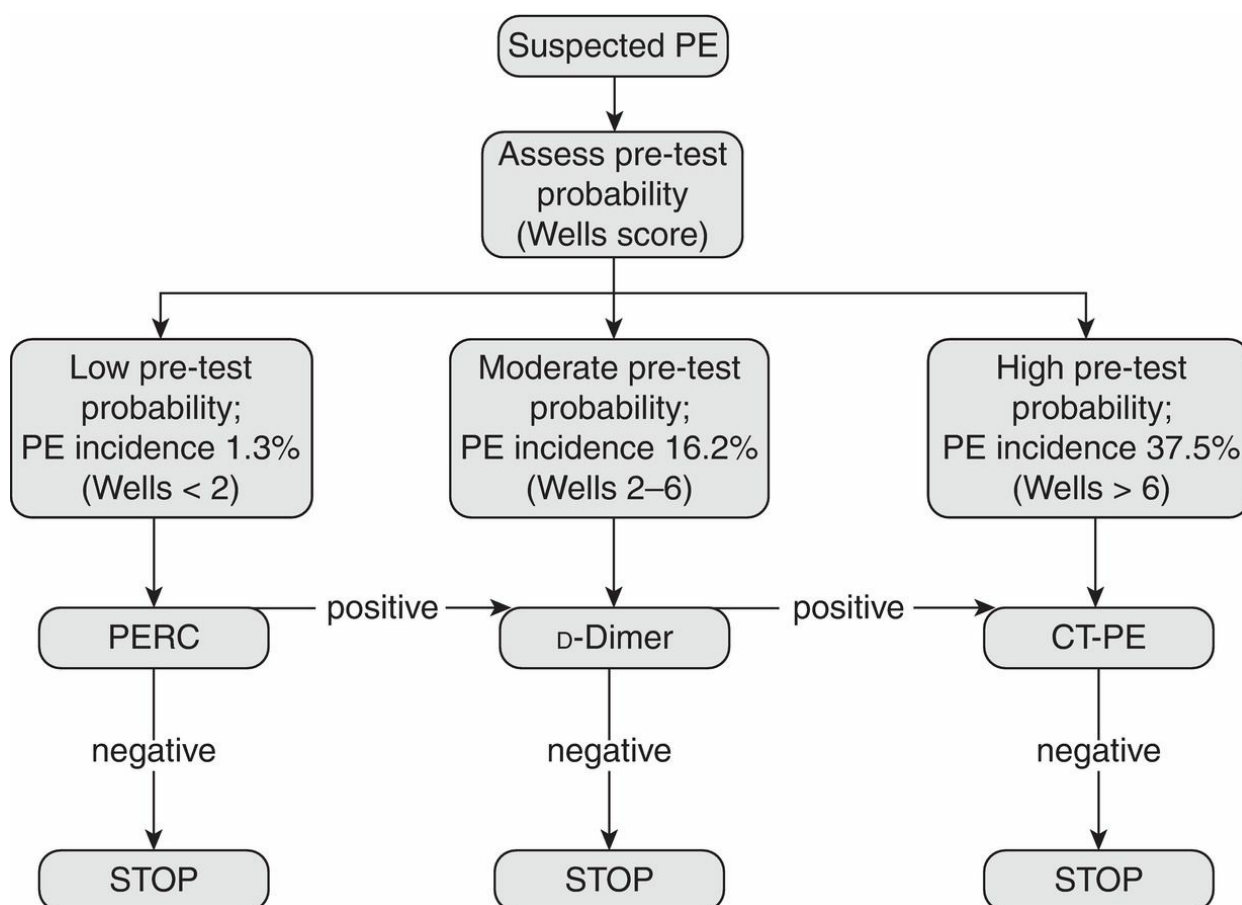
If a patient has a moderate pretest probability of PE or does not meet the PERC exclusion criteria in the low probability group, then it is appropriate to order a D-dimer assay. D-dimer is a degradation product of cross-linked fibrin that is elevated in PE because of the concurrent activation of coagulation and fibrinolysis. While the D-dimer has a high sensitivity and negative predictive value for PE, its specificity is poor as fibrin is also produced in other inflammatory states. The D-dimer should not be used in those patients with a high pretest probability of PE given its low negative predictive value in that population; a negative D-dimer may also not exclude PE in patients with symptoms over 14 days or those already on anticoagulation. As D-dimer concentration also typically increases with age, Douma and colleagues instituted an “age-adjusted” D-dimer cutoff value of (patient’s age × 10) µg/L. In patients aged 50 years or older with a low to moderate pretest probability and determined, this modified cutoff greatly increased the proportion of patients in whom PE could be safely excluded.

In patients with a high pretest probability or those in the low or moderate groups with a positive D-dimer, a computed tomography angiogram (CTA) of the chest is the next step in the diagnostic workup. The PIOPED II trial established that CTA has 83% sensitivity and 96% specificity for the diagnosis of PE. The addition of venous compression ultrasonography of the lower extremities does not significantly alter the posttest probability of PE in patients with a negative CTA, though may be helpful in proceeding to treatment without obtaining a CTA in certain patient populations, such as those whose renal function precludes contrast administration and in pregnant patients. Given the negligible utility of repeat CTA after an initial negative study, Kline and colleagues published a clinical decision rule recommending that patients with dyspnea and a normal CTA undergo echocardiography

given the high probability of isolated RV dysfunction or overload.

For those patients with a positive CTA, the pulmonary embolism severity index (PESI) can be applied to determine the risk of a 30-day mortality using 11 clinical criteria that are assigned varying point values: age, sex, history of cancer, history of heart failure, history of chronic lung disease, heart rate > 110 bpm, systolic blood pressure < 100 mm Hg, respiratory rate > 30 breaths per minute, temperature < 36°C, altered mental status, and oxygen saturation < 90%. For those patients with very low risk (score < 65), all studies showed a 30-day mortality <2% and low-risk (66 to 85) patients had a 90-day mortality of 1.1%. A noninferiority trial further demonstrated that very-low-risk and low-risk patients could have been treated as outpatients in the appropriate clinical setting.

The prevalence of confirmed PE in patients undergoing diagnostic workup is low (10% to 35%). It therefore becomes necessary to utilize these diagnostic strategies to achieve a balance between appropriate diagnosis and avoidance of unnecessary testing. A diagnostic algorithm is provided in [Figure 236.1](#).



**Figure 236.1** Risk stratification algorithm.

## KEY POINTS

- Determination of clinical pretest probability is the foundation of any diagnostic strategy and may be established by clinical gestalt or application of the Wells score.
- In patients with a low pretest probability of PE, one may apply the PERC rule. A PE can be ruled out if all eight criteria are present.
- If a patient has a moderate pretest probability of PE or does not meet the PERC exclusion criteria in the low probability group, order a D-dimer assay.
- In patients with a high pretest probability or those in the low to moderate groups with a positive D-dimer, a CTA is the next step in the diagnostic workup.
- PESI can be applied to determine the risk of 30-day mortality.

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# KNOW HOW TO DIAGNOSE AND TREAT PULMONARY EMBOLISM

**RYAN DICK-PEREZ, DO AND NICHOLAS M. MOHR, MD,  
MS, FACEP**

Pulmonary embolism (PE) is a potentially fatal disease that is frequently diagnosed in the emergency department. The clinical presentation of PE is often nonspecific, which makes the diagnosis elusive and easy to miss. Both missed diagnosis and delayed therapy can have devastating consequences on patient outcome. The severity of disease can range from a small peripheral PE that has no significant clinical effects to massive PE that leads to hemodynamic collapse and shock.

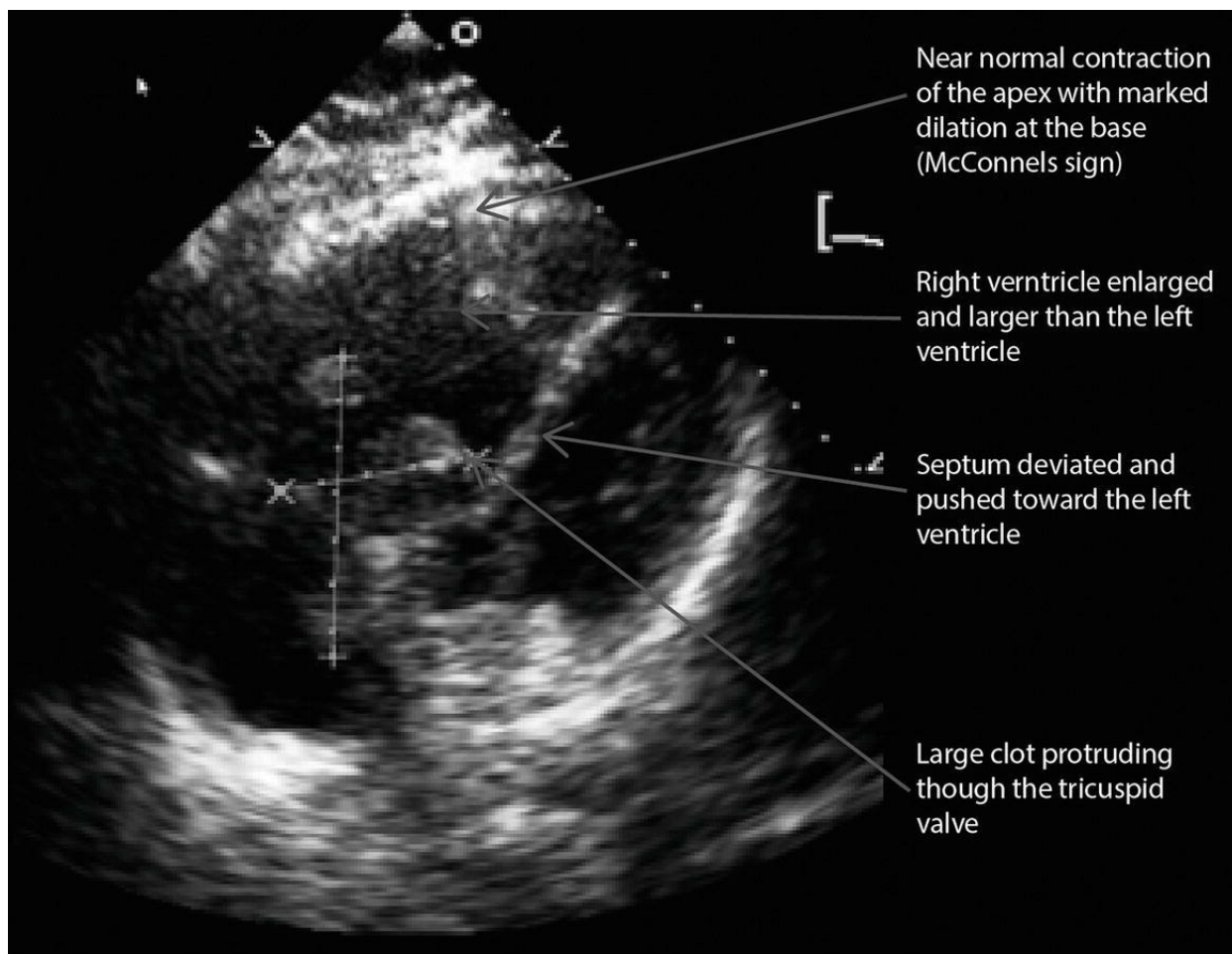
PE accounts for ~5% of all cardiac arrests and an even higher percentage of patients with pulseless electrical activity. Massive PE obstructs right ventricular outflow and causes right ventricular failure. Massive PE is defined as a PE that causes hypotension (systolic blood pressure <90 mm Hg for more than 15 minutes). Importantly, isolated tachycardia, hypoxemia, and radiographic or echocardiographic criteria alone are not sufficient to classify a PE as massive.

Bedside echocardiography is a rapidly available test that can be quickly performed and interpreted by emergency physicians. Right ventricular dysfunction is the finding most associated with acute PM. Additional findings of right ventricular dysfunction are described in [Table 237.1](#). In an unstable patient with a clinical scenario consistent with PE, these findings may be sufficient to proceed with treatment.

**TABLE 237.1 SIGNS CONSISTENT WITH MASSIVE OR SUBMASSIVE**

## PULMONARY EMBOLISM ON BEDSIDE ECHOCARDIOGRAPHY

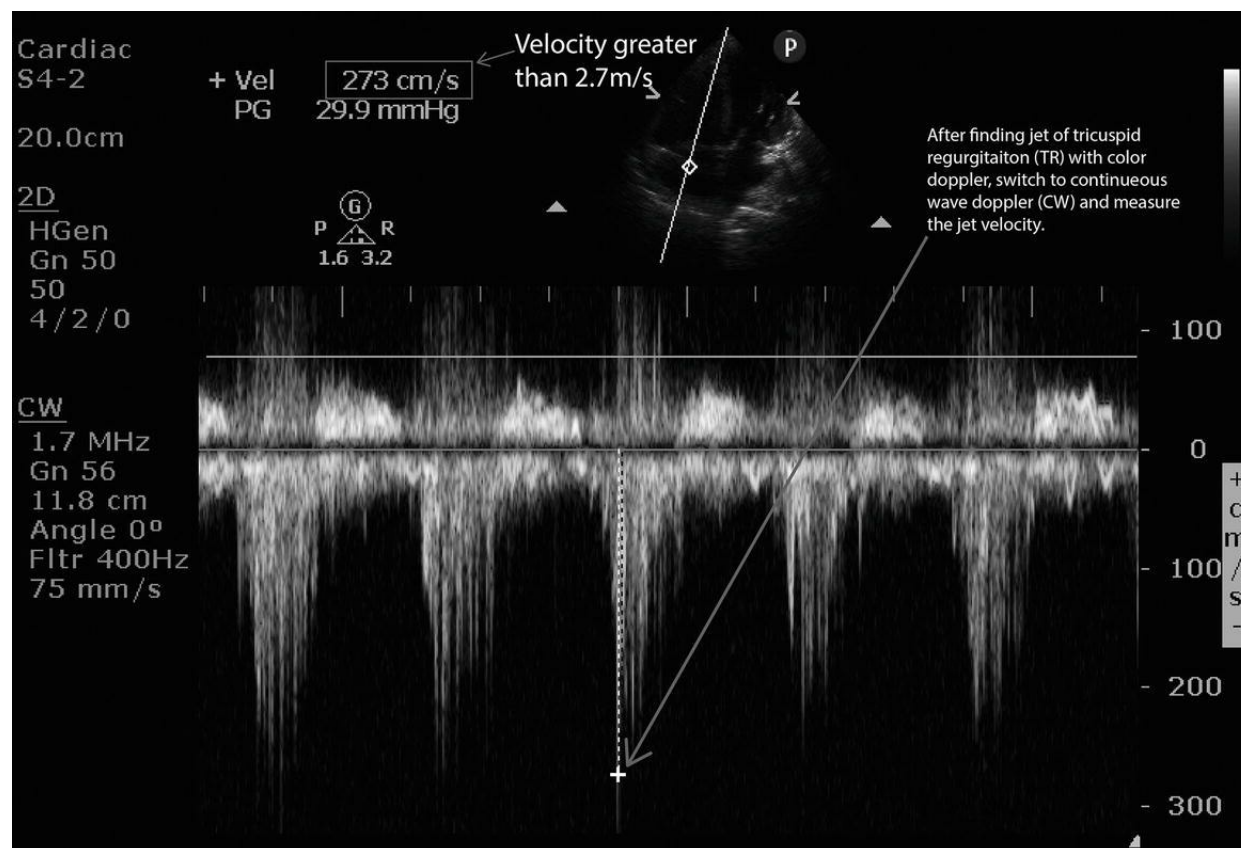
| FINDING  | DESCRIPTION  |
|--|--|
| McConnell sign   | Normal motion of the apex with akinesia of the septum and free wall  |
| Dilated right ventricle                                  | Right ventricle appears larger than left on apical or parasternal view ( <i>Figure 237.1</i> ).  |
| Paradoxical septal wall motion                           | A flattening of the septum or movement toward the left ventricle during systole, commonly described as the “D sign.” This occurs as a result of the increased right heart pressures overcoming the left heart pressures.             |
| Prolonged isovolumetric contraction and relaxation phase | When looking at both ventricles, a prolonged isovolumetric contraction and relaxation phase of just the RV indicates acute pulmonary hypertension, whereas prolongation in both ventricles indicates chronic pulmonary hypertension. |
| Increased tricuspid regurgitation (TR)                   | TR velocity of $>2.7$ m/s ( <i>Figure 237.2</i> )  |
| Thrombus seen in the right ventricle                     | A hyperechoic structure within the right ventricle that is often ill defined and mobile  |



**Figure 237.1** Subxiphoid 4-chamber view with echocardiographic evidence of pulmonary embolism with right heart strain, including



right ventricular dilation, McConnell sign, septal deviation, and apparent residual clot in the right ventricle.



**Figure 237.2** Pulmonary artery pressures can be estimated echocardiographically, and correspond to the degree of ventricular strain in acute pulmonary embolism. In this continuous wave Doppler measurement of the tricuspid regurgitation jet, the tricuspid gradient is estimated at 29.9 mm Hg based on a peak velocity of 2.7 m/s. Adding this estimate to the concurrently measured central venous pressure of 11 mm Hg yields an estimated systolic pulmonary artery pressure of 40.9 mm Hg.

PE treatment is aimed at (1) preventing further embolization and (2) reducing the pulmonary clot burden. Initially, systemic anticoagulation is used to decrease the rate of clot propagation. In high-risk patients, anticoagulation may be initiated prior to definitive diagnosis. Initial anticoagulant agents include low-molecular-weight heparin, fondaparinux, or unfractionated heparin (best for renal insufficiency, high bleeding risk).

Traditionally, all patients with a PE have been admitted for anticoagulation and monitoring. More recent data suggest that low-risk patients with a PE may be discharged from the emergency department with



injectable anticoagulants or non–vitamin K oral anticoagulants. Low-risk PE is defined according to select stratification criteria, such as the simplified pulmonary embolism severity index (sPESI). The sPESI score is listed in [Table 237.2](#). Those with a sPESI score of 0 and reliable follow-up can be discharged. Patients who do not meet criteria for low-risk PE should be admitted.

**TABLE 237.2 SIMPLIFIED PULMONARY EMBOLISM SEVERITY INDEX**

| <b>RISK FACTOR</b>                         | <b>POINT</b> |
|--|--------------|
| Age > 80 years old                         | 1            |
| Cancer                                     | 1            |
| Chronic heart failure or pulmonary disease | 1            |
| Heart rate > 100                           | 1            |
| Systolic blood pressure < 100 mm Hg        | 1            |
| Oxygen saturation < 90%                    | 1            |
| Score of 0 has a 1.1% risk of death        |              |

For unstable patients with PE, thrombolytic therapy should be considered. Thrombolytic therapy has been shown to decrease mortality in patients with massive PE who are at low risk for bleeding. The American College of Chest Physicians recommends the administration of 100 mg of alteplase over 2 hours. Importantly, the rate of intracranial hemorrhage in this setting is ~2%. Thrombectomy should be considered in patients who fail thrombolysis or have contraindications to this therapy. Improved outcomes have been observed when thrombectomy is performed early in the course of management. For centers that do not have the capability to perform open thrombectomy, catheter-based techniques should be considered.

Hemodynamic support for patients with massive PE can be challenging. Large volume fluid resuscitation will further dilate the right ventricle and may worsen hemodynamics. Patients often require both vasopressor and inotropic medications. Norepinephrine is the vasopressor agent of choice for massive PE, while inotropic support can be provided with epinephrine, dobutamine, or milrinone. For patients who continue to deteriorate despite medical therapy, extracorporeal membrane oxygenation (ECMO) can be considered at centers with the capability to perform this resource-intensive therapy. ECMO is generally reserved for cases of refractory shock, refractory hypoxemia, or cardiac arrest.

The management of patients with submassive, or intermediate, PE is discussed in [Chapter 238](#).

## KEY POINTS

- Bedside echocardiography can help confirm the diagnosis of PE in critically ill patients.
- Overzealous fluid administration can increase right ventricular dilatation and worsen hemodynamics.
- Patients with massive PE should generally be treated with a thrombolytic agent.
- Norepinephrine is the vasopressor of choice in patients with a PE.
- Mechanical clot retrieval, direct intra-arterial thrombolytic administration, and ECMO are rescue therapies that can be used in PE patients with refractory shock.

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# KNOW WHICH PATIENTS WITH SUBMASSIVE PULMONARY EMBOLISM MAY BENEFIT FROM THROMBOLYTIC THERAPY

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**SANGEETH DUBBIREDDI, MD AND LILLIAN L. EMLET,  
MD, MS**

For patients with massive pulmonary embolism (PE) and low-risk PE, the treatment options are straightforward. For massive PE, the severe afterload on the right ventricle (RV) must be emergently unburdened either with thrombolysis or surgical embolectomy. For hemodynamically stable patients with a PE and no evidence of RV dysfunction, standard anticoagulation is adequate. For patients with submassive PE (also referred to as intermediate risk), defined as hemodynamically stable with evidence of RV dysfunction, the selection of the right therapeutic intervention remains controversial. For this group of patients, the benefits of thrombolysis (i.e., decrease in death, pulmonary hypertension, and recurrent PE) may be offset by the risk of major bleeding events, such as intracerebral hemorrhage (ICH).

For patients with a newly diagnosed PE, evidence of RV dysfunction can be detected on the electrocardiogram (ECG), computed tomography angiogram (c) of the chest, echocardiography (ECHO), and biochemical markers. Classic ECG abnormalities associated with PE include an incomplete or complete right bundle-branch block, T-wave inversions in lead V<sub>1</sub> to lead V<sub>4</sub>, and the combination of an S wave in lead I, Q wave in lead III, and T-wave inversion in lead III (S1Q3T3). RV dysfunction is indicated on CTA of the chest if the ratio of the internal diameter of the RV to the left

ventricle is  $>0.9$  in the transverse plane. On ECHO, RV dysfunction is defined as any one of following findings:

- RV end-diastolic diameter  $>30$  mm (parasternal long-axis or short-axis view)
- Right-to-left ventricular end-diastolic diameter  $> 0.9$  (apical or subcostal 4-chamber view)
- Hypokinesis of the right ventricular free wall (any view)
- Tricuspid systolic velocity  $>2.6$  m/s (apical or subcostal 4-chamber view)

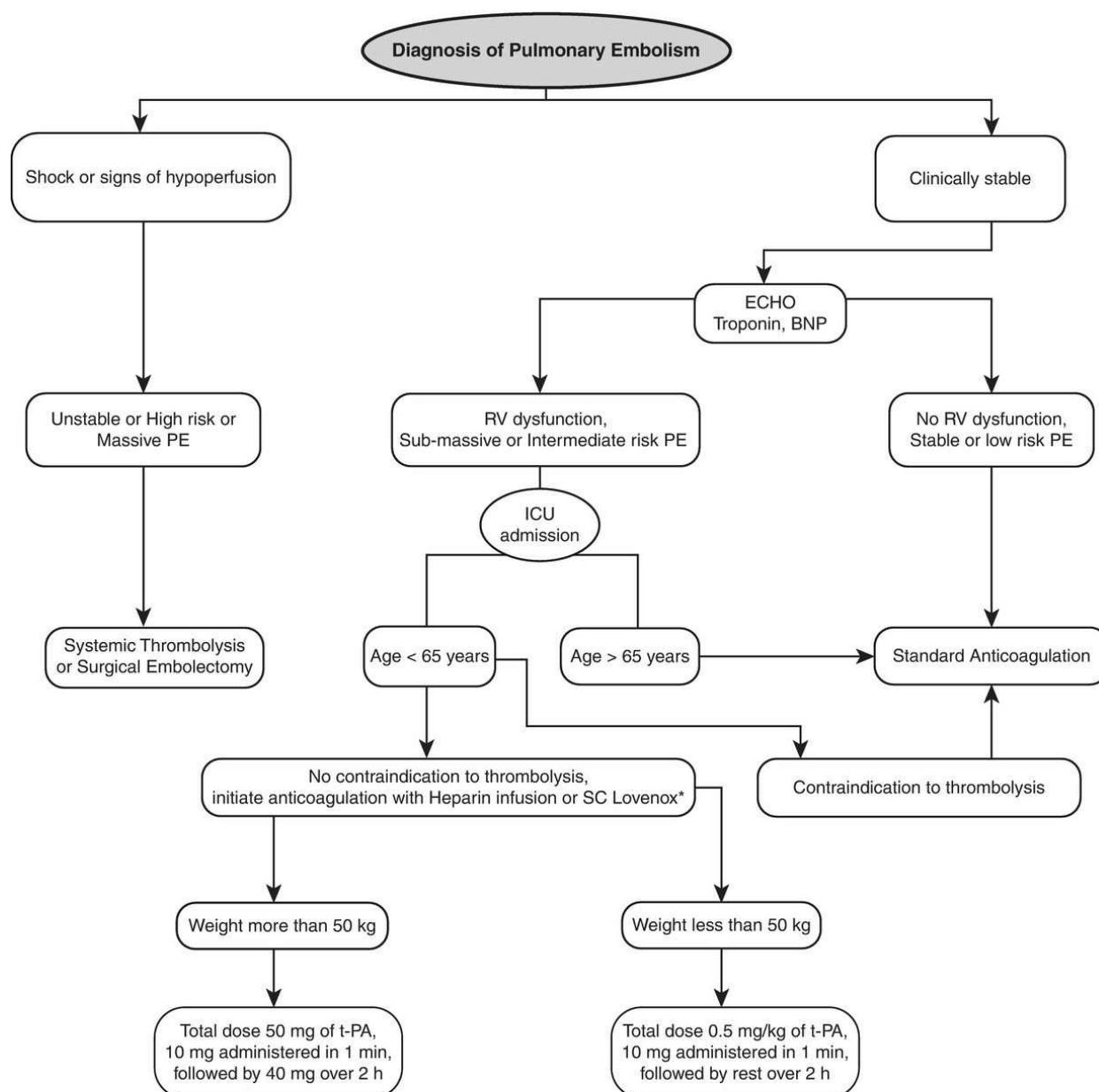
Biochemical markers of RV dysfunction include the following lab abnormalities:

- Brain natriuretic peptide (BNP)  $> 90$  pg/mL or NT-proBNP  $> 900$  pg/mL
- Troponin I  $> 0.06$   $\mu\text{g/L}$  or troponin T  $> 0.01$   $\mu\text{g/L}$

The use of systemic thrombolysis in patients with a submassive PE might be considered for clinically hemodynamically stable individuals who have objective evidence of RV dysfunction (as detailed above) without absolute contraindication for thrombolysis. However, the evidence for systemic thrombolysis, even in this specific patient population, remains uncertain because of the lack of standardization on type, dose, or route of thrombolytic administration used in recent studies. Equally important is the timing of initiating anticoagulation after administration of the thrombolytic agent. For patients older than 65 years of age, current data suggest that for every 51 patients with a submassive PE treated with thrombolysis, 1 person is saved. In this age group, major bleeding occurs in 1 out of every 176 patients treated with thrombolysis. Major bleeding in various trials is loosely defined as intracranial hemorrhage, a decrease in hemoglobin of 2 g/dL within 24 hours that requires a transfusion, or the need for endoscopic, radiologic, or surgical intervention. For patients older than 65 years of age, the risk of bleeding with thrombolysis is too high to provide therapeutic benefit. This is comparable to aspirin where the number needed to treat is 42 for an ST-segment elevation myocardial infarction (STEMI), while the number needed to harm for nondangerous bleeding is 167.

Thrombolytic medications can also be administered via a catheter that is placed into the pulmonary arteries using ultrasound guidance. While recent studies have demonstrated the benefit of a lower dose of thrombolytic therapy with ultrasound guidance when compared to anticoagulation alone, it has not been compared to systemic administration of thrombolytic agents.

Because the pulmonary circulation receives 100% of the cardiac output in comparison with cerebral or coronary circulations, it can be hypothesized that the dose required to lyse the clot may not need to be as high as that for a stroke or a STEMI nor should it necessitate a special catheter to deliver it to the pulmonary circulation to mitigate risk of bleeding. Until further trials are done, the optimal dose of thrombolytic therapy for patients with submassive PE is one that causes the least harm and provides the most economic benefit. [Figure 238.1](#) illustrates a PE algorithm.



\* If no renal dysfunction, use Enoxaparin 1 mg/kg SC, maximum dose not to exceed 80 mg BID. If renal dysfunction, use IV Heparin infusion with initial bolus at 70 mg/kg, max dose not to exceed 6,000 Units. During t-PA Infusion and 3 hours after infusion, heparin infusion was dosed at 10 U/kg/h or a maximum of 1,000 U/hr. Subsequent dose titrated to 1.5–2 times aPTT with initial maintenance of 18 U/kg/h.

Doses adapted from the MOPETT trial.

**Figure 238.1** Decision making algorithm for pulmonary embolism.

## KEY POINTS

- Submassive PE is defined as hemodynamically stable with evidence of RV dysfunction.
- RV dysfunction can be detected on ECG, CTA of the chest, ECHO, BNP, or troponin.
- The number needed to treat with thrombolytic therapy is 51 for

patients older than 65 years of age with a submassive PE.

- The risk of bleeding with thrombolysis outweighs the benefit in patients older than 65 years of age.
- The optimal dose of thrombolytic therapy for patients with submassive PE is one that causes the least harm and provides the most economic benefit.

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# UNDERSTAND PROPER VENTILATOR MANAGEMENT IN PATIENTS WITH ACUTE ASTHMA EXACERBATIONS

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Acute severe asthma, or status asthmaticus, refers to an episode of bronchoconstriction that is unresponsive to standard management and can rapidly progress to respiratory failure. Patients with acute severe asthma will present with significant respiratory distress, and it is critical to rapidly treat them to avoid significant morbidity and mortality. Maximal medical management of these patients includes inhaled bronchodilators, intravenous fluids, epinephrine, and possibly noninvasive positive pressure ventilation. Intubation and mechanical ventilation should not be taken lightly. Invasive ventilation of the asthmatic patient can be fraught with peril and lead to further increase in morbidity and mortality.

The initial management of acute severe asthma should focus first on breathing, then circulation, and finally the airway (“BCA”). When patients fail to respond to medical therapy, intubation and mechanical ventilation may be required. It is important to recall that patients with severe asthma have significant limitations with exhalation. Bronchoconstriction makes it difficult for them to completely exhale their inspired tidal volume. In addition, tachypnea shortens the time during which they can exhale. These issues cause positive end-expiratory pressure (PEEP) to accumulate, which leads to dynamic hyperinflation. As these patients get sicker, they develop carbon dioxide (CO<sub>2</sub>) retention and respiratory acidosis.

When a patient is intubated and placed on a mechanical ventilator,



ventilation converts from negative pressure to positive pressure. This leads to increased intrathoracic pressures, which can compromise venous return and right ventricular function and cause hypotension. A common pitfall in ventilating the asthmatic patient is to set the ventilator to “blow off” CO<sub>2</sub> in an attempt to resolve the respiratory acidosis. This will worsen hyperinflation and cause barotrauma at the level of the alveoli. As intrathoracic pressures rise, the risk of developing a tension pneumothorax also increases.

A critical concept in ventilating the asthmatic patient is to use a low respiratory rate. A low respiratory rate will provide the patient enough time to fully exhale each administered breath and avoid dynamic hyperinflation. A downside to the use of low respiratory rates is the accumulation of CO<sub>2</sub>, a concept known as permissive hypercapnea. Arterial concentrations of CO<sub>2</sub> are allowed to rise to supranormal levels in order to avoid breath stacking. As a result, pH should be closely monitored to avoid dangerous acidosis. It is also important to note that the tidal volume is based on ideal body weight and not actual body weight. Using the actual body weight can lead to the administration of unnecessarily large volumes and worsen hyperinflation. If the use of low respiratory rates does not result in adequate time for exhalation, the inspiratory flow time can be increased. This will increase the time spent in the expiratory phase. Initial ventilator settings for the asthmatic patient are listed in [Table 239.1](#).

**TABLE 239.1 INITIAL VENTILATOR SETTINGS**

- Respiratory rate: 6–8 breaths/min
- Tidal volume: 6–8 mL/kg (ideal body weight)
- PEEP: 0–5 cm H<sub>2</sub>O
- FiO<sub>2</sub>: minimum necessary to maintain oxygen saturation >93%
- Inspiratory flow rate: 100–120 L/min

Ventilator alarms should be immediately addressed. Most ventilators are set to alarm for increased peak pressures. Peak pressures reflect the pressures endured in large airways and within the ventilator tubing, but do not reflect the pressures sensed in the alveoli. Because asthmatic patients may have very high inspiratory flow rates, the peak pressures will typically be elevated. A better measure of barotrauma and the potential for alveolar damage is called plateau pressure. Plateau pressure can be checked by pressing the inspiratory pause button on the ventilator and should be maintained below 30 cm H<sub>2</sub>O.

If the plateau pressure is higher than 30 cm H<sub>2</sub>O, the alveoli are susceptible to barotrauma and adjustments to the ventilator setting should be made to decrease this pressure. This can be achieved by decreasing the respiratory rate further followed by reductions in the tidal volume. These adjustments, in turn, will lead to increased arterial concentrations of CO<sub>2</sub> and acidemia.

Despite utilizing the permissive hypercapnea approach to ventilation, asthmatic patients can still develop hemodynamic instability and hypoxia after intubation. Rapid recognition of the underlying problem and initiation of treatment are critical and can be achieved using the DOPES mnemonic. This is listed in [Table 239.2](#).

**TABLE 239.2 DOPES MNEMONIC**

- Displacement of the endotracheal tube (ETT)
  - Action: Direct visualization with laryngoscopy
- Obstruction of the endotracheal tube
  - Action: Pass ETT suction catheter
- Pneumothorax (tension)
  - Action: Lung ultrasound for lung sliding
  - Action: Empiric needle decompression or finger thoracostomy
- Equipment failure
  - Action: Disconnect the vent and deliver manual BVM breaths
- Stacked Breaths
  - Action: Gently push down on anterior chest wall with two hands until no further exhalation detected

## KEY POINTS

- Invasive ventilation of the asthmatic patient can be fraught with peril and lead to further increases in morbidity and mortality.
- A common pitfall in ventilating the asthmatic patient is to set the ventilator to “blow off” CO<sub>2</sub> in an attempt to resolve the respiratory acidosis.
- A critical concept in ventilating the asthmatic patient is to use a low respiratory rate.
- Tidal volume is based on ideal body weight and not actual body weight.
- If the use of low respiratory rates does not result in adequate time for exhalation, the inspiratory flow time can be increased.

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# KNOW THE CAUSES, EVALUATION, AND MANAGEMENT OF HEMOPTYSIS

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Hemoptysis is the expectoration of blood from a subglottic source. Massive hemoptysis accounts for ~5% to 15% of cases and is often described as more than 600 mL of blood in a 24-hour period. Since the majority of patients do not wait 24 hours to present to the emergency department (ED) after expectorating blood, a more practical definition for massive hemoptysis is the expectoration of more than 100 mL/h or a volume of blood sufficient to impair gas exchange or cause hemodynamic instability. Importantly, the alveoli may contain up to 400 mL of blood before gas exchange is impaired. Pitfalls in the evaluation of hemoptysis include the failure to identify the source of bleeding (pulmonary versus gastrointestinal), failure to appreciate the danger posed by the cause (i.e., pulmonary embolism, bioterrorism agents), and an underestimation of the volume or rate of hemorrhage.

## CAUSES

Accurate determination of the precise etiology of hemoptysis is often not possible in the ED; however, knowledge of the most common causes of hemoptysis permits appropriate disposition and management. The most common causes are listed in [Table 240.1](#). Bronchitis, bronchiectasis, pneumonia, and tuberculosis account for up to 80% of cases of hemoptysis. Bronchiectasis, pneumonia, bronchogenic carcinoma, and tuberculosis are the most common causes of massive hemoptysis. The sources of bleeding in up to 90% of cases of massive hemoptysis are the bronchial arteries, with the

remaining 10% of cases from pulmonary or systemic arteries.

**TABLE 240.1 CAUSES OF HEMOPTYSIS**

## **Bioterrorism agents**

- Anthrax
- Plague
- Tularemia

## **Cardiac disease**

- Congenital heart disease
- Congestive heart failure
- Endocarditis
- Mitral stenosis

## **Chronic lung conditions**

- Bronchiectasis
- Chronic obstructive pulmonary disease

## **Coagulopathy or platelet dysfunction (drug-induced anticoagulants or thrombolytics or endogenous coagulopathies)**

## **Infectious**

- Bronchitis
- Fungal or parasitic infection
- Lung abscess
- Pneumonia (HIV, cytomegalovirus, herpes simplex virus, *Legionella*, aspergillosis, hantavirus, leptospirosis, *Mycoplasma*, other bacteria)
- Tuberculosis

## **Inflammatory/autoimmune disease**

- Goodpasture disease
- Systemic lupus erythematosus
- Wegener granulomatosis
- Henoch-Schönlein purpura, thrombotic thrombocytopenic purpura, immune thrombocytopenic purpura
- Connective tissue disorders
- Antiphospholipid antibody syndrome, cryoglobulinemia

## **Malignancy (primary or metastatic)**

## **Toxins**

- Cocaine abuse
- Nitrogen dioxide inhalation
- Drugs (propylthiouracil, amiodarone, methotrexate, haldol, nitrofurantoin, sirolimus, bleomycin)

## **Trauma**

- Blunt or penetrating chest trauma
- Foreign body aspiration
- High-altitude pulmonary edema
- Iatrogenic

## **Vascular**

- Bronchovascular fistula
- Collagen vascular disease
- Dieulafoy lesion, pulmonary arteriovenous malformation
- Pulmonary embolism

## **Catamenial hemoptysis (pulmonary endometriosis)**

## **Cryptogenic (no cause identified despite thorough evaluation, up to 1/3 of patients)**

## **Pseudohemoptysis (upper gastrointestinal or upper airway source of bleeding)**

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## EVALUATION

The history of present illness should focus on the identification of risk factors for the most common conditions. This includes a history of tobacco use, vasculitis, immunosuppression, venous thromboembolism, and tuberculosis. It is also important to ask about a history of coagulopathy and the use of antiplatelet or anticoagulant medications. Exposure to toxins or a recent history of chest trauma is also important to note. Attempts should be made to quantify the volume of blood expectorated and describe its composition (i.e., gross blood, clots, blood-streaked sputum).

Key findings on physical examination include the presence of petechiae or ecchymosis, a diastolic murmur consistent with mitral stenosis, a new murmur concerning for endocarditis, asymmetric lung sounds, or asymmetric extremity edema that suggests deep venous thrombosis. The oral and nasal cavities should be inspected for an upper airway source of bleeding. If the history is insufficient to exclude hematemesis, a nasogastric aspirate can be tested for blood. In addition, the bloody material produced by the patient may be pH tested, with an acidic pH indicative of a gastrointestinal source and an alkaline pH suggestive of a pulmonary source.

A chest x-ray (CXR) will be abnormal in more than 50% of patients with hemoptysis. Stable patients with a nondiagnostic CXR should undergo computed tomography (CT) of the chest with intravenous contrast. Results of the CT will determine the need for bronchoscopy as well as subsequent management. Laboratory tests depend on the condition of the patient and may include markers of bleeding severity and respiratory failure (i.e., hemoglobin, arterial blood gas), studies to diagnose the cause (i.e., coagulation studies, sputum Gram stain, acid-fast bacillus, D-dimer), as well as tests to facilitate treatment (i.e., type and cross).

## MANAGEMENT

Patients with massive hemoptysis are ideally managed at a facility with access to interventional pulmonology (for balloon tamponade, topical hemostatic application, or iced saline lavage), interventional radiology (for bronchial artery embolization), and thoracic surgery (for lobectomy or pneumonectomy if other measures fail). Arrangements for hospital transfer should be made when it is anticipated that the patient may need these services. Unstable patients require immediate treatment to optimize oxygenation and ventilation and avoid asphyxiation from blood. Blood

product transfusion to correct anemia, optimize hemodynamics, and correct coagulopathy and platelet dysfunction should be pursued early. If the side of bleeding can be determined from the history, exam, or imaging, the patient should be placed in a bleeding-lung-down position in order to exploit gravity and keep blood from spilling into the nonhemorrhagic lung.

In cases of massive hemoptysis that result in respiratory failure, endotracheal or endobronchial intubation should be performed. Use of a size 8.0 tube or larger is desirable to facilitate clot removal and introduction of a flexible bronchoscope. If the side of bleeding has been determined and the bleeding is life threatening, the unaffected lung may be isolated by intubation of that main bronchus. This is ideally attempted with bronchoscopic guidance. Intubation of the right main bronchus is complicated by occlusion of the right upper lobe bronchus, but is often the quickest and safest solution for left lung hemorrhage. If performed blindly, rotation of the endotracheal tube 90 degrees in the direction of the desired side can aide in correct placement. If this fails, placement of a double-lumen tube or use of bronchial blockers may be considered when available.

The majority of ED patients will have “nonmassive” hemoptysis. Most patients with <~30 mL of blood in 24 hours can be discharged, provided they have normal vital signs, a stable ED course, and no significant comorbid conditions or acute life-threatening conditions. Most importantly, timely referral and follow-up should be arranged.

## KEY POINTS

- Carefully inspect the oral and nasal cavities to evaluate for an upper airway source of bleeding.
- Consider nasogastric aspirate and pH testing of the material to differentiate hematemesis from hemoptysis.
- Patients with massive hemoptysis should ultimately be managed at a facility with interventional pulmonologists, interventional radiologists, and thoracic surgeons.
- If identified, the bleeding lung should be placed in a dependent position.
- In cases of life-threatening massive hemoptysis, intubation of the main bronchus of the nonbleeding lung with a size 8.0 or larger tube should be attempted.



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# USE HIGH-FLOW NASAL CANNULA IN PATIENTS WITH MILD TO MODERATE RESPIRATORY DISTRESS FROM HYPOXEMIA

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**ROSS McCORMACK, MD AND JONATHAN ELMER, MD,  
MS**

Emergency providers often classify patients with respiratory distress into select categories based on severity of illness. Patients with mild to moderate respiratory distress generally have the ability to phonate and do not demonstrate significant hypoxemia, as measured by pulse oximetry. In contrast, patients with severe respiratory distress may have significant hypoxemia, hypercarbia and altered mental status, or signs of imminent respiratory failure (i.e., severe tachypnea, cyanosis). Some patients may even present with a mixture of hypoxemia and hypercarbia. High-flow nasal cannula (HFNC) has emerged as a potential therapy for patients with mild to moderate hypoxemic respiratory failure.

HFNC devices can deliver up to 60 L/min of heated, humidified oxygen to an adult patient. In contrast to the traditional nonrebreather mask that entrains room air through side-port holes, HFNC devices can deliver a fraction of inspired oxygen ( $\text{FiO}_2$ ) close to 100% because of flow rates that exceed the patient's intrinsic peak inspiratory flow. HFNC devices may also provide a small amount of positive end-expiratory pressure (PEEP), though this remains controversial. Several studies have suggested that HFNC devices can generate ~5 to 8 cm  $\text{H}_2\text{O}$  of PEEP. The amount of PEEP

generated is dependent upon nasal prong position and whether the patient's mouth is open or closed. Unfortunately, PEEP cannot truly be measured in these devices as it can with traditional noninvasive ventilation (NIV) devices (i.e., continuous positive airway pressure, bilevel positive airway pressure). Additional benefits of HFNC devices include the ability to decrease dead space in the upper airway through a washout of carbon dioxide and improved minute ventilation. Finally, the heated and humidified air improves patient tolerance of the device and may enhance mucociliary clearance.

At present, the best evidence for the use of HFNC is in the patient with isolated hypoxemia due to pneumonia that does not require immediate airway management and mechanical ventilation. A prospective, multicenter, randomized, controlled trial in patients with hypoxemic respiratory failure and normal work of breathing demonstrated decreased mortality with the use of HFNC compared with NIV. Importantly, mortality was a secondary outcome, and the benefit was primarily seen in patients with pneumonia. Patients with chronic lung disease were excluded from this trial. HFNC may also be used in patients with hypoxemia due to other etiologies (i.e., congestive heart failure); however, the evidence that supports its use is less robust.

The use of HFNC for patients with hypercapnic respiratory failure is limited to case reports. It is important to note that HFNC devices do not have a direct effect on tidal volume or respiratory rate, the two primary determinants of ventilation and carbon dioxide exchange. Risks of HFNC use in this specific patient population include the administration of high levels of oxygen that may mask worsened pulmonary function and delayed intubation. As a result, the use of HFNC in patients with hypercapnic respiratory failure cannot be routinely recommended.

HFNC has also been used for preoxygenation and to provide apneic oxygenation for patients undergoing rapid sequence intubation. Most notably, this has been used in the critically ill obese patient. However, there is currently no study that has formally demonstrated the benefit of HFNC in the setting of preoxygenation compared to a nonrebreather mask with nasal cannula.

Given the size of the device and lack of robust clinical data, the use of HFNC in this setting should not be considered standard care. If HFNC is used to preoxygenate select patients prior to endotracheal intubation, consider using a jaw-thrust maneuver to maintain a patent airway and maximize its effect.

## KEY POINTS

- HFNC delivers heated, humidified oxygen at a high concentration and washes out dead space from the upper airway.
- Consider HFNC in patients with mild to moderate respiratory distress secondary to hypoxemic failure.
- The ideal clinical situation for HFNC may be patients with pneumonia and hypoxemia.
- HFNC should not be routinely used for patients with hypercapnic respiratory failure. NIV is a better choice.
- At present, HFNC does not appear to be more effective for preoxygenation prior to intubation compared to a nonrebreather mask with a standard nasal cannula at 15 L/min.

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# SECTION XVIII

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## TOX

# ALCOHOL INTOXICATION AND WITHDRAWAL

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CANDICE JORDAN, MD

## ALCOHOL INTOXICATION

Habitual users of alcohol are seen in the emergency department (ED) on an almost daily basis. Though most patients with alcohol intoxication can be managed without any medical intervention, there are important disease states present in those with history of alcohol abuse that cannot be missed.

Hypoglycemia is commonly seen in patients with history of alcohol abuse due to malnourishment and inhibition of gluconeogenesis. This should be identified quickly and treated with oral glucose if the patient's mental status allows or with IV dextrose if needed. Additionally, these patients are at high risk for vitamin deficiencies, most notably thiamine deficiency. Wernicke encephalopathy is the neurologic manifestation of thiamine deficiency and is estimated to be present in 0.2% to 3% of patients, but is missed in 75% to 85% of cases. Diagnosis is challenging, as many symptoms resemble those of acute alcohol intoxication including gait ataxia, confusion, nystagmus, and bowel/bladder dysfunction. There are no adjunct diagnostic tests or imaging to guide ED diagnosis of Wernicke's, and therefore, diagnosis must be made on a clinical basis. The classic triad of ophthalmoplegia, ataxia, and confusion is rarely seen. Left untreated, Wernicke's may lead to Korsakoff syndrome, which is an untreatable form of dementia and portends significant morbidity and mortality. Treatment of Wernicke's is high-dose parenteral thiamine, though the duration of therapy remains debated. Due to the high rates of thiamine deficiency, it is recommended that all patients with a history of alcohol abuse disorder should be treated prophylactically with thiamine. Fortunately, thiamine is a very safe

supplement with no overdose syndrome and an almost absent side effect profile.

The classic teaching is that thiamine should be given before glucose to prevent the precipitation of Wernicke's. In fact, the basis of this theory is multiple case series and reports, and there is no clear evidence that this is true. However, if your patient requires treatment for hypoglycemia, it should be followed promptly by thiamine supplementation.

Clinicians should also be aware that patients with history of alcohol abuse are at increased risk for "occult trauma." In addition to a thorough physical exam, a low threshold should be reserved for further evaluation of the patient who is assumed to be intoxicated.

## **ALCOHOL WITHDRAWAL**

At the other end of the spectrum is alcohol withdrawal. An estimated 40% of all patients who abuse alcohol will develop an acute alcohol withdrawal syndrome (AWS) if they abruptly stop or substantially reduce their alcohol intake. Alcohol withdrawal is a clinical spectrum that is characterized by autonomic hyperactivity after abrupt discontinuation of alcohol in patients who have developed a physical dependence.

The pathophysiology of alcohol withdrawal is complex. It is thought that chronic alcohol use induces CNS neurotransmitter remodeling, particularly down-regulation of the inhibitory GABA receptors and up-regulation of excitatory glutamatergic receptors. Abrupt cessation of alcohol results in an imbalance of neurotransmitter activity and CNS hyperexcitability.

The diagnosis of alcohol withdrawal is on the basis of history and physical. It is very important to determine the precipitating cause of alcohol withdrawal, as you do not want to miss an underlying infection or injury that may have prompted alcohol cessation. Providers must maintain a high degree of clinical suspicion for alcohol withdrawal in patients who are critically ill or have a depressed level of consciousness. There are many well-validated tools for assessing the presence and severity of alcohol withdrawal including CIWA, AWS, and PAWSS.

First-degree withdrawal symptoms typically onset 6 to 12 hours after the last drink. Patients may exhibit tremors, diaphoresis, nausea, vomiting, hypertension, and tachycardia. About 12 to 24 hours after the last drink, patients may develop visual and tactile visual hallucinations with an otherwise clear sensorium. Approximately 10% of patients with withdrawal symptoms will go on to develop withdrawal seizures—typically generalized tonic-clonic seizures with little or no postictal period. Delirium tremens

(DTs) represents the most severe manifestation of acute alcohol withdrawal and carries a very high mortality if untreated. It usually occurs 48 to 72 hours after the last drink, but may not appear until many days later. Symptoms include disorientation, delirium, hyperthermia, seizures, and agitation and may last for 5 to 7 days even with therapy. Older patients, those with prior history of DT, and those with history of heavier drinking are at the highest risk of developing DT. Alcohol withdrawal is a continuous spectrum of symptoms, but it is important to remember that not all patients follow the same clinical course. AWS may start with mild symptoms and become progressively worse or can start with DT.

The goal of therapy is to minimize the severity of symptoms and to prevent progression to severe symptoms. Not all patients require medical therapy or admission. Initial interventions should include decreasing stimulation including providing reassurance and putting the patient in a dark, calm area. Benzodiazepines represent the pharmacologic gold standard for the treatment of alcohol withdrawal and, to date, are the only medications proven to prevent symptomatic worsening of alcohol withdrawal. The choice of benzodiazepine varies widely based on your clinical practice, and all are effective. There is greater evidence for longer-acting benzodiazepines (diazepam, chlorthalidone) as they produce a smoother withdrawal. However, in elderly patients or those with advanced liver disease, the use of shorter-acting agents may decrease risk of oversedation. Patients with mild withdrawal symptoms can likely be treated with oral formulations, whereas those with moderate to severe symptoms should be treated with IV medications. Symptom-triggered therapy is superior to fixed-dose therapy, and there is no limit to the quantity of benzodiazepines that can be administered to a patient with AWS in carefully monitored settings. Escalation of therapy may be warranted for those patients requiring very high doses of benzos and continue to have worsening symptoms. In these rarer cases, barbiturates may augment the effects of benzos. Additionally, propofol is another therapeutic option in patients with refractory symptoms.

## KEY POINTS

- Remember to do a thorough physical exam and check a dexi (bedside finger stick glucose level) on “intoxicated patients” with alterations of their mental status. Consider giving a dose of prophylactic parenteral thiamine.
- Be sure to determine why alcohol was abruptly discontinued—you do not want to miss an underlying infection or injury!



- Alcohol withdrawal symptoms do not always present in order—frequently reassess patients to determine need for escalated therapy.
- There is no limit to the quantity of benzodiazepines that you can give to treat alcohol withdrawal symptoms.

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# **ACETAMINOPHEN TOXICITY: GETTING REACQUAINTED WITH MATTHEW AND RUMACK**

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**DAVID ROSE, MD**

The most common cause of acute liver failure in the United States is acetaminophen (APAP) poisoning. Toxicity may result from intentional or unintentional ingestions, either acutely or chronically. Acute toxicity begins to occur with ingestions of >150 mg/kg in the pediatric population or more than 7.5 to 10 g in the adult population in an 8-hour period. Peak serum APAP concentrations occur at about 4 hours post ingestion. APAP is metabolized mostly by conjugation with sulfate and glucuronide and is excreted in the urine. A small percentage is oxidized by the cytochrome P450 system to toxic *N*-acetyl-*p*-benzoquinone imine (NAPQI), which is normally conjugated with glutathione. If this system is overwhelmed, liver injury via excess NAPQI can occur. The antidote for APAP poisoning is *N*-acetylcysteine (NAC), which acts to replete glutathione and thus reduces the toxic effects of NAPQI via conjugation.

Avoid common errors in acetaminophen toxicity by assuring the following items as noted below.

## **START NAC EMPIRICALLY IN CERTAIN LIVER FAILURE PATIENTS**

If there is concern for APAP poisoning with any signs or symptoms of liver failure then treat with NAC. Administration of NAC has been shown to decrease morbidity and mortality in APAP-induced liver failure regardless of

time line.

## **IF THE APAP LEVEL IS BELOW THE TREATMENT LINE THEN DO NOT START NAC**

In an acute overdose, obtain a serum APAP level 4 hours after ingestion. The Rumack-Matthew Nomogram predicts the risk of hepatotoxicity based on time line and APAP level. The nomogram can only be used between 4 and 24 hours post ingestion. Use the modified Rumack-Matthew Nomogram where the 4-hour “treatment line” intersects at 150 mcg/mL; APAP levels above this call for treatment with NAC, those below do not. The original line begins at 200 mcg/mL at 4 hours. The more conservative “treatment line” is plotted 25% below the original line (150 mcg/mL), allows for a margin of error and is recommended by the Food and Drug Administration. The original line separated people with and without elevated aminotransferases taking APAP. It is not a line separating patients with and without hepatic failure, further increasing the treatment lines’ sensitivity for those who do and do not need treatment. This increased safety gives confidence not to treat with levels below this line. If the APAP level is above the 150 mcg/mL line, treat with NAC.

With sustained release products, there is some uncertainty on the safest approach. While some authorities suggest only one APAP level, it may be prudent to consider a subsequent APAP level at 4 to 6 hours when extended release products are involved. It should be noted that the nomogram was designed and validated for a single APAP value. Nomogram “line crossing” can occur with subsequent levels but has never been shown to affect outcome.

If unsure of time of ingestion, use the earliest possible point in time. This allows for the most conservative treatment approach. If no time can be estimated or the time window is >24 hours, check APAP and aspartate aminotransferase (AST) levels and start NAC if either one is elevated. If both levels are normal, then treatment with NAC is not indicated.

## **START NAC EMPIRICALLY IF APAP LEVEL WILL RESULT AFTER 8 HOURS**

If APAP level cannot be obtained within 6 to 8 hours of ingestion, then administer NAC using history of APAP ingestion alone while awaiting laboratory values. NAC is most effective when administered within 8 hours

of ingestion, and thus, empiric treatment is beneficial until therapy can be guided with objective data and the Rumack-Matthew nomogram.

## **TREAT CHRONIC INGESTIONS BASED ON HISTORY AND APAP/AST LEVELS**

Patients at risk for chronic APAP poisoning include those with alcohol abuse, febrile children, and patients using P450-inducing medications.

If chronic toxicity is suspected (10 g or 200 mg/kg in 24 hours; or 6 g a day or 150 mg/kg/d in 48 hours), then get APAP and AST levels. If either value is elevated, then start NAC. Of course, consider other possible causes of liver failure.

## **ORDER AN APAP LEVEL IN PATIENTS WITH ANY SUSPECTED INGESTION**

In 1996, Sporer and Khayam-Bashi published a study out of San Francisco General Hospital, which found that with universal screening, 0.3% of all suspected ingestions (suicide attempts and altered mental status) had a potentially toxic APAP levels that would not have been discovered based on history alone. Because APAP toxicity is common and the consequences of not making the diagnosis is deadly, check an APAP level in any patient with altered mental status or intentional overdose. Remember that APAP is found in a multitude of medications.

### **KEY POINTS**

- Start NAC empirically in liver failure, when APAP toxicity is suspected.
- If the APAP level is below the treatment line, then do not start NAC.
- Start NAC empirically if APAP level will result after 8 hours.
- Treat chronic ingestions based on history and APAP/AST levels.
- Order an APAP level in patients with any suspected ingestion.

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## MIXED DISTURBANCE: THINK SALICYLATE POISONING

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**HARRY E. HEVERLING, DO AND TIFFANY C. FONG, MD**

Salicylates are among the oldest known medications, with the recorded use of salicylate containing willow bark by ancient Sumerians and Egyptians. In 1899, scientists at Bayer isolated acetylsalicylic acid and sold the compound as aspirin.

Salicylates have a variety of medical applications and are commonly used for their analgesic, antipyretic, and anti-inflammatory properties. They are found in numerous formulations in addition to the commonly used acetylsalicylic acid; these include bismuth salicylate in antidiarrheal medications (Kaopectate and Pepto-Bismol), salicylic acid keratolytics for acne, and methyl salicylate containing topical ointments and liniments (Icy Hot, Bengay, oil of wintergreen, and some Chinese herbal preparations). Notably, many liniments can be highly concentrated with salicylates, which can lead to severe poisoning if ingested.

Salicylate toxicity is important to recognize, due to the ubiquity of salicylates, the numerous preparations in which they are found, and their morbidity and mortality in overdose. The therapeutic range of serum salicylate is 10 to 20 mg/dL. Clinical signs and symptoms of toxicity are observed at serum concentrations above 30 mg/dL (associated with salicylate ingestion >150 mg/kg). Early clinical manifestations of toxicity include tinnitus, nausea, vomiting, diarrhea, and fever.

Salicylate toxicity has a complex pathophysiology affecting multiple organ systems. A mixed acid-base disturbance is highly characteristic of salicylate poisoning, beginning with a primary respiratory alkalosis. This is mediated by stimulation of the medullary respiratory center causing

hyperventilation, manifested as both tachypnea and hyperpnea. Through a number of mechanisms, a superimposed anion gap metabolic acidosis follows.

The metabolic acidosis causes shifting of salicylate into a nonionized state, which enables passage across the blood-brain barrier into the central nervous system (CNS). The CNS is severely impacted by salicylate toxicity. Tinnitus may progress to hearing loss and deafness, vertigo may develop, and CNS dysfunction may progress to delirium, agitation, and lethargy, followed by seizures and coma. Pulmonary manifestations of acute lung injury may also occur. Hyperthermia, due to uncoupling of oxidative phosphorylation, is an ominous sign.

Many signs and symptoms of salicylate poisoning may be confused with other critical illnesses. An index of suspicion must be maintained, as delayed diagnosis and management of severe salicylate toxicity may have mortality as high as 15%. Treatment should be initiated as soon as possible, with goals of reducing CNS exposure to salicylate, optimizing salicylate elimination, and correcting fluid and electrolyte abnormalities.

In patients with normal mental status, multiple dose-activated charcoal should be given as early as possible to reduce absorption following ingestion. The recommended dose is a 10:1 ratio of activated charcoal to ingested salicylate.

Essential to the management of salicylate poisoning is alkalinization with sodium bicarbonate. Serum alkalinization shifts salicylate molecules to an ionized form, thus excluding additional entry into and facilitating distribution out of the CNS. Urine alkalization enhances elimination of salicylate by several mechanisms, including trapping the ionized salicylate in the urine for excretion. Alkalinization should be considered in patients with an elevated serum salicylate concentration with clinical symptoms. Alkalinization is achieved with a 1 to 2 mEq/kg IV bolus of sodium bicarbonate followed by a continuous infusion. The sodium bicarbonate infusion is prepared through mixture of 3 ampules (150 mEq) of sodium bicarbonate in 1 L of 5% dextrose in water (D5W). This is run at 1.5 to 2 times the maintenance fluid rate and titrated to a goal serum pH of 7.45 to 7.55 and goal urine pH of 7.50 to 8.0.

Intravenous fluid resuscitation is also usually necessary, as patients are hypovolemic due to vomiting, fever, and hyperventilation (insensible losses). Glucose and other electrolytes should be repleted as necessary. Administration of glucose is important due to discordance between measured serum glucose and cerebrospinal fluid glucose levels. Special attention should be paid to the correction of hypokalemia. In the setting of

hypokalemia, the renal tubules reabsorb potassium ions in exchange for hydrogen ions, thus hindering the efforts to alkalinize urine.

While endotracheal intubation and mechanical ventilation may be necessary in critically ill patients with deteriorating mental status, respiratory fatigue or acute lung injury, the utmost vigilance must be maintained to prevent worsening acidemia from respiratory acidosis. Ventilator settings must maintain a minute ventilation similar to the patient's preintubation ventilatory status (i.e., hyperventilation). Acidosis can be worsened by sedation prior to intubation, and it must be minimized with IV boluses of sodium bicarbonate and hyperventilation with bag-mask ventilation.

Hemodialysis is indicated in cases of severe salicylate poisoning, to remove salicylate from the serum and more easily correct acid-base, fluid, and electrolyte abnormalities. Indications for dialysis include worsening clinical status despite aggressive supportive measures, persistent CNS disturbance, acute lung injury, renal insufficiency, severe acid-base or electrolyte disturbance, or serum salicylate levels greater than 100 mg/dL.

## KEY POINTS

- Salicylates are found in many formulations including oral and topical analgesics, antidiarrheal medications, and acne treatments.
- Tinnitus, hyperventilation, and altered mental status or other neurologic dysfunction should heighten suspicion for salicylate poisoning. Hyperthermia, due to uncoupling of oxidative phosphorylation, is an ominous sign.
- Salicylate toxicity has a hallmark acid-base disturbance, with a mixed primary respiratory alkalosis and anion gap metabolic acidosis.
- Alkalinization with sodium bicarbonate reduces CNS exposure to salicylate and enhances salicylate elimination.
- Endotracheal intubation in a severe salicylate poisoning is a high-risk procedure due to potential to produce life-threatening acidosis from respiratory suppression.
- Hemodialysis resources should be mobilized early for severe salicylate poisoning.

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## TOXIC ALCOHOLS

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**CANDICE JORDAN, MD**

Methanol, ethylene glycol, and isopropanol are toxic alcohols that when ingested may cause significant morbidity. Toxic alcohol intoxication is a topic well covered in medical education, but in practice is not quite so easy to recognize and treat. It requires a high index of suspicion by the treating clinician and appropriate interpretation of laboratory data. Acute ingestion of all alcohols may cause inebriation, depending on the quantity ingested. The alcohols themselves are not acutely toxic, but their metabolites can be fatal.

The alcohols are all oxidized in the liver by alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH) to their toxic metabolites. Ethanol, the most commonly ingested alcohol, also binds ADH, but with a nearly 10 to 20 times greater affinity than the other alcohols. Therefore, coingestion ethanol delays the metabolism of the other alcohols thereby delaying onset of acidosis and symptoms and in some cases may protect the patient from some of the toxic effects.

Methanol is best known for its use in windshield wiper fluid, but can also be found in model car fuel, solid cooking fuel, and even in colognes and perfumes. Methanol is most often ingested, but there are case reports of methanol toxicity due to inhalational and transdermal exposure. Patients typically begin to experience symptoms of methanol toxicity 12 to 24 hours after significant ingestion. Methanol is metabolized to formic acid, which is toxic to the retina, optic nerve, and brain parenchyma. Patients will complain of visual disturbances, GI upset, and dyspnea and may develop irreversible blindness, even despite appropriate therapy.

Ethylene glycol's primary use is as antifreeze for car radiators. Its sweet taste and fluorescent blue color makes it a target for unintentional ingestion by children. Patients typically develop symptoms 6 to 12 hours after ingestion. Ethylene glycol is metabolized to glycolic acid (which causes the

acidosis) and later to oxalic acid (which causes the toxic effects). Oxalic acid forms a complex with calcium and precipitates in the renal tubules leading to acute kidney injury and even renal failure. In addition, these patients may develop seizures, coma, and cardiovascular failure. Many formulations of antifreeze contain fluorescein (to detect radiator leaks), and the urine of some who has ingested ethylene glycol may fluoresce with a wood's lamp if examined within 6 hours.

Isopropanol is different than the other toxic alcohols in that it does not cause acidosis and tends to be less toxic than the other alcohols. Better known as rubbing alcohol, isopropyl alcohol is widely found in the household for its medicinal uses and is also used in various cosmetic and pharmaceutical products including hand sanitizer. It is metabolized to acetone, which causes ketosis, but not acidosis. Clinical features of isopropanol ingestion include CNS depression ranging from lethargy to coma, respiratory depression and even seizures. Isopropanol is a direct irritant to mucosal surfaces and may cause nausea, vomiting, and severe hemorrhagic gastritis.

At this time, rapid testing for toxic alcohol levels or their metabolites is not widely available and therefore cannot guide need for therapy. Therefore, other surrogate markers of toxic alcohol ingestion must be used to make the diagnosis. Methanol, ethylene glycol, and isopropanol are osmotically active agents, which cause an elevated osmol gap. It is important to remember that osmol gaps vary from patient to patient and over time. In addition, in order to accurately calculate the osmol gap, a metabolic panel and ethanol level must be drawn at the same times as the serum osmolality. There is controversy over what osmol gap is concerning, as there are many conditions that cause an elevated gap. An osmol gap  $>15$  is elevated, and one  $>50$  is highly concerning for toxic alcohol ingestion. Over time, as the alcohol is metabolized to its acid metabolite, the osmol gap will decrease, and the anion gap will increase causing a severe metabolic gap acidosis. Isopropanol will not cause an anion gap acidosis.

The mainstay of therapy for methanol and ethylene glycol ingestions is ADH inhibition with fomepizole. Isopropanol ingestions can be treated with supportive care, as its metabolite is not toxic. Fomepizole is a competitive inhibitor of ADH activity with an affinity nearly 1,000 times that of the other toxic alcohols and prevents formation of toxic metabolites after toxic alcohol ingestion and has been shown to decrease the need for hemodialysis (HD). It can be administered as a bolus every 12 hours and does not require monitoring of serum concentration. Theoretically, ethanol could also be used as a therapy for toxic alcohol poisoning as it also has a greater affinity for ADH. However, fomepizole is far superior in that it binds ADH with greater

affinity and has many fewer side effects than ethanol.

Any patient with a plausible history of ingestion or with a markedly elevated osmol or anion gap of unclear etiology should be treated until alcohol concentrations are available.

HD is the definitive therapy as it clears both the toxic alcohol and its metabolite. HD should be initiated in patients with end-organ damage, severe acidosis, and acute renal failure.

## KEY POINTS

- Remember to check an osmol gap in patients who present with early toxic alcohol exposure.
- Isopropanol causes an elevated osmol gap, but not an elevated anion gap.
- Coingestion with ethanol may lead to a delay in symptoms and acidosis
- Late presentation of methanol and ethylene glycol alcohol ingestion can have a normal osmol gap, but an elevated anion gap metabolic acidosis.
- Early treatment with fomepizole may prevent need for HD.

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# **THE FIVE STAGES OF IRON TOXICITY: BEWARE OF THE LATENT PERIOD**

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**CHRISTINA CLARK, PA-C**

Iron supplementation is commonly used in the outpatient setting for patients with iron deficiency anemia, in prenatal vitamins for patients who are pregnant, and in over-the-counter multivitamins. Iron supplementation can be found in three forms, ferrous gluconate, ferrous sulfate, and ferrous fumarate, each of which contains a varying amount of elemental iron. Iron toxicity can occur when large doses are consumed, whether intentionally or unintentionally. Generally, symptoms of iron toxicity will begin to occur once doses of 40 to 60 mg/kg have been reached. After toxic levels have been consumed, clinical manifestations of iron toxicity can present in five stages; however, the stages can often overlap. Therefore, the practitioner should be vigilant of the deteriorating patient.

The first stage of iron toxicity can present 30 minutes to 6 hours after ingestion and typically include generalized gastrointestinal (GI) complaints including nausea, vomiting, diarrhea, and abdominal pain. The patient may also develop gastrointestinal bleeding with hematemesis or melena.

The second stage of iron toxicity can occur 6 to 24 hours after ingestion. Signs and symptoms may include lethargy, tachycardia, hypotension, hypovolemia, and metabolic acidosis. The practitioner must stay observant during this stage as patients with low grade toxicity and mild gastrointestinal symptoms may be resolving; however, patients with higher levels of toxicity may progress and rapidly deteriorate into the next stage.

The third stage of iron toxicity can present as early as 6 hours after

ingestion up to 72 hours after ingestion and is marked by cardiovascular toxicity and collapse. The patient will progress into hypovolemic, distributive, or cardiogenic shock. Anion gap metabolic acidosis, coagulopathy, hepatic dysfunction, renal failure, acute respiratory distress syndrome (ARDS) and coma may develop and contribute to mortality during this stage of toxicity.

The fourth stage of iron toxicity is typically seen 2 to 4 days after ingestion and manifests as severe hepatic failure and necrosis. Due to hepatic dysfunction, hypoglycemia may occur. Hepatic failure contributes to mortality during this phase of toxicity.

The fifth and final stage of iron toxicity will present 2 to 8 weeks after initial ingestion. The most common clinical presentation will be bowel obstruction or stricture secondary to gastrointestinal scarring.

In addition to the early identification of iron toxicity based on history and physical exam, diagnosis can be assisted through results of laboratory and imaging results. Standard laboratory studies include a full chemistry panel, hepatic profile, complete blood count, arterial or venous blood gas, coagulation studies, lactate, type and cross-match, pregnancy test if appropriate, and serum iron concentration. Serum iron concentration levels peak 4 to 6 hours after ingestion (and at 8 hours for slow-release iron formulations). An abdominal x-ray may confirm ingestion if radiopaque tablets are present but cannot rule out ingestion if absent on film.

Serum iron concentration levels may correlate with toxicity at the following levels:

- <300 mcg/dL: minimal GI symptoms
- 350 to 500 mcg/dL: mild to moderate GI symptoms
- >500 mcg/dL: iron toxicity
- >1,000 mcg/dL: severe morbidity and mortality, hepatic failure

Patients with iron toxicity should receive close hemodynamic monitoring, early supportive care, and aggressive fluid resuscitation. Orogastric lavage with a large-bore orogastric tube should be considered to assist in removal of iron tablets visualized on abdominal radiograph; however, iron tablets may be too bulky to be removed via orogastric tube. Therefore, whole bowel irrigation should be initiated for patients with large amounts of iron tablets identified on abdominal radiograph.

The antidote for severe iron toxicity is chelation therapy with intravenous deferoxamine. Initial dosing of deferoxamine is 15 mg/kg/h with recommended titration upwards to a maximum dosing of 35 mg/kg/h.

Though typical length of therapy with deferoxamine is 24 hours, a phone consultation with your local poison control center is recommended and may be accessed via the national Poison Help Line at 1-800-222-1222. The emergency provider should acknowledge possible adverse effects of deferoxamine including hypotension with rapid or high-dose IV infusions, ARDS with prolonged infusion times, and sepsis, as both iron toxicity as well as deferoxamine infusion may predispose individuals to *Yersinia enterocolitica*. Additionally, a gastroenterology and critical care medicine consult is recommended.

Disposition recommendations for patients with iron toxicity in the emergency department include the following:

- Ingestion of 10 to 20 mg/kg may be observed for 6 to 12 hours; patients with minimal or no GI manifestations can be discharged home with close follow-up.
- Ingestion of 20 to 60 mg/kg should be hospitalized for observation.
- Hemodynamic instability, lethargy, shock, or metabolic acidosis should be admitted to the intensive care unit.

## KEY POINTS

- Iron supplementation is common.
- Toxic doses of iron occur at doses of about 40 mg/kg.
- There are five stages of iron toxicity, including a quiescent latent period that occurs after about 6 hours.
- Careful hemodynamic monitoring and fluid resuscitation should be considered in all cases of iron toxicity.
- Consider whole bowel irrigation and chelation therapy with deferoxamine.

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# DON'T MISS ANTICHOLINERGIC SYNDROMES!

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THEODORE FAGRELIUS, MD

The anticholinergic toxidrome can occur with a wide variety of prescription and OTC medications as well as many plants and mushrooms. The most common drugs with anticholinergic activity are antihistamines, antipsychotics, antispasmodics, skeletal muscle relaxants, and tricyclic antidepressants. The most common plants and mushrooms containing anticholinergic alkaloids include jimsonweed (*Datura stramonium*), deadly nightshade (*Atropa belladonna*), and fly agaric/amanita mushrooms (*Amanita muscaria*). Anticholinergic agents competitively antagonize the effects of the neurotransmitter acetylcholine at muscarinic receptors.

It is important to make the distinction between the central anticholinergic syndrome, which is the alteration of mental status due to CNS muscarinic blockade, and the peripheral anticholinergic syndrome, which is the antagonism of muscarinic receptors outside the CNS, which appears clinically as a “blockade” of the parasympathetic nervous system. Although the central and peripheral characteristics of the anticholinergic syndrome typically occur concomitantly, the central signs and symptoms may occur with minimal or no peripheral ones.

Central muscarinic antagonism produces a variety of neuropsychiatric manifestations (“mad as a hatter”), including anxiety, agitation, confusion, visual hallucinations, bizarre behavior, delirium, coma, and seizures. One of the earliest and most frequently identified hallmarks of peripheral anticholinergic toxicity is tachycardia, which is due to the effects of decreased vagal tone on the atrioventricular node. The other important common sign of peripheral anticholinergic poisoning is anhydrosis (“dry as a bone”). Other common signs included hyperthermia and compensatory

peripheral vasodilatation (“hot as a hare” and “red as a beet”), mydriasis (“blind as a bat”), and urinary retention (“full as a flask”). Gastrointestinal disturbances (ileus) are common as well and decreased gastrointestinal motility could result in delayed absorption of the toxic agents, thus prolonging their effects.

The diagnosis must be made clinically based on a history of exposure and the presence of characteristic signs and symptoms as specific drug levels are generally not available and many tests are nonspecific. Electrocardiogram monitoring and laboratory studies including electrolytes, glucose, CPK, and blood gases will often be useful in making the diagnosis. The typical presentation of the anticholinergic syndrome can often be mistaken for a wide variety of etiologies, and therefore, the diagnostic workup begins broadly. Many medical conditions and drugs can cause signs and symptoms consistent with anticholinergic toxicity. Furthermore, many classes of drugs and toxins have anticholinergic effects; therefore, one must differentiate pure anticholinergic toxicity from poisonings in which anticholinergic toxicity is but one aspect. Physostigmine should be considered as both a diagnostic test and a therapeutic measure in the setting of central anticholinergic syndrome; a trial dose can be used to confirm the presence of anticholinergic toxicity. Rapid reversal of the abnormal mental status is consistent with the diagnosis.

Emergency and supportive measures include the maintenance of an open airway and assisting ventilation as needed. Decontamination with activated charcoal may be administered in appropriate conditions in which the patient is alert and cooperative and the charcoal can be administered within roughly 1 hour of the ingestion. The anticholinergic toxidrome is often treated with supportive measures (fluids, cooling, etc.), benzodiazepines, and physostigmine.

For specific drug and antidote treatment, a small dose of physostigmine (a reversible inhibitor of acetylcholinesterase, which increases the synaptic concentration of acetylcholine to overcome the toxic receptor blockade) can be given to patients with severe anticholinergic toxicity (e.g., hyperthermia, severe delirium, and tachycardia). However, most patients will do well with supportive care alone. The dose for physostigmine is 0.5 to 1.0 mg slow IV push over 2 to 5 minutes, which may be repeated with 0.5 mg increments up to a total of 2 mg over the first hour. The onset of action is within 3 to 8 minutes, the duration of effect is 30 to 90 minutes, and the elimination half-life is 15 to 40 minutes. Caution must be used with physostigmine as it can cause cardiac conduction disturbances, bradyarrhythmias, and asystole, especially in patients with exposures to antipsychotics, a TCA overdose, or a prolonged QRS > 100. Physostigmine should also be used cautiously in patients with reactive airway disease, intestinal obstruction, and a seizure

history. Also, physostigmine should not be used with concurrent use of depolarizing neuromuscular blockers (e.g., succinylcholine). The patient should be on cardiac monitors when physostigmine is administered.

## KEY POINTS

- Anticholinergic toxicity is common and should definitely be considered in the differential diagnosis of patients with altered mental status or with a history of any ingestion.
- Although the central and peripheral characteristics of the anticholinergic syndrome typically occur concomitantly, the central signs and symptoms may occur with minimal or no peripheral ones.
- Physical examination should evaluate the degree of sweating in a patient with a suspected toxidrome as anhydrosis is a characteristic of anticholinergic toxicity in contradistinction to diaphoresis, which is commonly seen in the similarly appearing sympathomimetic syndrome.
- Determine the presence or absence contraindications for physostigmine and use it as a diagnostic and therapeutic measure.
- Consultation with the regional Poison Center can help with diagnosis and treatment plans and should be considered prior to administration of physostigmine.

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## CHOLINERGIC POISONING

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**THEODORE FAGRELIUS, MD**

The cholinergic toxidrome is produced by an excess of the neurotransmitter acetylcholine (ACh), which results in overstimulation of muscarinic and nicotinic receptors. This syndrome can be caused by acetylcholinesterase inhibitors (e.g., medicines such as neostigmine, rivastigmine, and physostigmine; pesticides; and nerve gas), muscarinic agents (e.g., bethanechol, pilocarpine, and mushrooms), and nicotinic agents (e.g., nicotine). Organophosphate (OP) and carbamate containing pesticides cause the cholinergic toxidrome. Carbamate refers nonspecifically to a chemical compound that is also present in many acetylcholinesterase inhibitor medicines. The syndrome of cholinergic toxicity includes a wide variety of causes, but it is most often observed in the setting of pesticide (OP or carbamate variety) poisonings.

The initial presentation of a patient with a cholinergic toxidrome can vary widely based on the individual toxin's rate of action, the route of absorption, lipid distribution, and its metabolism. The diagnosis is made clinically, and a history of exposure or ingestion to the toxin is critical (e.g., Intentional or accidental ingestion of a medicine? Exposure to pesticides? Nerve gas attack?). Inhibition of acetylcholinesterase leads to increased synaptic ACh at both central and peripheral muscarinic and nicotinic cholinergic receptor sites. There are both nicotinic and muscarinic receptors in the brain that contribute to the signs and symptoms of respiratory depression, lethargy, seizures, and coma. Peripherally, muscarinic receptors are located at effector organs of the parasympathetic system. Stimulation causes bradycardia, miosis, sweating, hyperperistalsis, abdominal cramps, nausea, bronchorrhea, wheezing, excessive salivation, urinary incontinence, and seizures. Stimulation of nicotinic receptors at autonomic ganglia activates both parasympathetic and sympathetic systems, with unpredictable results. This often results in muscle fasciculations, cramping, tremor, tremor,

hypertension, and mydriasis. Excessive stimulation frequency can actually cause depolarization blockage, as in the case of succinylcholine, leading to weakness and paralysis. Thus, initial tachycardia and hypertension may be followed by bradycardia and heart block, and muscle fasciculations may be followed by paralysis. Combined with increased secretions and bronchospasm, respiratory muscle weakness leads to death by respiratory failure.

Treatment focuses on aggressive airway protection, liberal use of atropine for control of excessive airway secretions, decontamination, and, in the case of OP compounds, early administration of the antidote pralidoxime (2-PAM), which reactivates cholinesterase. Prompt recognition of toxicity and early intervention usually result in complete recovery.

Treatment should be directed at controlling secretions and respiratory status. Atropine should be administered intravenously at a dose of 2 to 5 mg (pediatric dose, 0.05 mg/kg) every 3 to 5 minutes, with the end point being control of respiratory secretions. Upper airway obstruction from vomit and secretions may occur, and bronchospasm and weakness can rapidly cause respiratory failure; thus, early intubation is important. Do not use succinylcholine for rapid sequence intubation in the patient poisoned with an OP because the effect will be extended secondary to the inhibition of the cholinesterase. Use a nondepolarizing agent instead. Tachycardia is not a contraindication to atropine administration and will likely occur when atropine is given. If tachycardia is significant, the rate of administration of atropine can be slowed. Mild poisonings may resolve with just 1 to 2 mg of atropine, and severe poisonings may require more than 1,000 mg. Large doses of atropine may lead to antimuscarinic CNS toxicity, and if such toxicity occurs, glycopyrrolate (1 to 2 mg; pediatric dose, 0.025 mg/kg) can be used in place of atropine.

Decontamination, when applicable, is an important aspect of management. Contaminated clothes should be removed immediately, and areas of topical exposure must be washed aggressively. Bowel decontamination, via gastric lavage or activated charcoal (<1 hour since ingestion, 1 g/kg up to a maximum of 50 g), is a potential treatment adjunct in appropriate patients, though neither is uniformly recommended.

2-PAM is the antidote for OP insecticide poisoning and should be given early based on the suspicion of cholinesterase poisoning. Although its efficacy may vary according to the structure of the OP compound, it should be given to all OP-poisoned patients. It works by increasing the rate of AChE regeneration by displacing the OP. It is a common belief that 2-PAM is not beneficial if given after 24 hours because of the “aging” of AChE. However,

OP insecticides have been detected in blood weeks after exposure because of their redistribution to fat. Therefore, late 2-PAM therapy may still be of benefit. The adult dose is 1 to 2 g via the intravenous route delivered over a 15- to 30-minute period followed by a continuous infusion of 500 mg/h. Pediatric dosing consists of a 25- to 50-mg/kg load followed by a 10- to 20-mg/kg/h infusion. 2-PAM is not indicated for carbamate poisoning, which is usually mild and self-limited, because carbamates do not irreversibly bind to the acetylcholinesterase molecule and therefore 2-PAM is not necessary. In the unknown patient suspected of suffering from a pesticide poisoning with cholinergic symptoms, 2-PAM should be administered before determining the causative agent. Because true cholinesterase regenerates at a rate of only 1% a day and the redistribution of the toxins in fat, it can take months for symptoms to resolve if cholinesterase is not regenerated with 2-PAM.

## KEY POINTS

- A mnemonic for cholinergic overdose is “SLUDGE and the Triple Bs” for salivation, lacrimation, urination, defecation, GI hypermotility, emesis, bronchorrhea, bronchospasm, and bradycardia.
- The initial treatment is 2 mg of atropine and 2 mg of 2-PAM.
- The testing of cholinesterase values is generally not useful in the emergency department for diagnosing OP toxicity. There is great variation in the values of both diseased and nondiseased patients.
- Carbamate poisoning is less severe than OP poisoning and CNS symptoms are uncommon because, unlike the OPs, these agents do not cross the blood-brain barrier and they are only transient cholinesterase inhibitors.
- Up to 40% of OP-poisoned patients will develop a neurological disorder within days to weeks of exposure. Characteristic findings include muscle weakness, decreased deep tendon reflexes, polyneuropathy, cranial nerve abnormalities, and respiratory muscle weakness.

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## **AN OLD FAVORITE HEART MEDICATION: DIGOXIN**

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**DANIEL B. SAVAGE, MD, MPH**

Digoxin is a cardiac glycoside used to treat heart failure and supraventricular cardiac arrhythmias such as atrial fibrillation and atrial flutter. While this medication is not frequently prescribed, its narrow therapeutic window leaves patients susceptible to both acute and chronic toxicity.

Digoxin works through inhibition of the cardiac Na/K ATPase pump leading to increased intracellular sodium and subsequent calcium influx through the Na/Ca pump causing increased myocardial contractility—which is useful in heart failure! Digoxin also suppresses AV nodal conduction, increasing the refractory period and decreasing rapid ventricular rates due to atrial arrhythmias.

Due to its mechanism of action, the primary clinical presentation of digoxin toxicity is cardiac arrhythmias, followed by electrolyte abnormalities (hyperkalemia in acute overdose, hypokalemia in chronic overdose), gastrointestinal distress (i.e., nausea/vomiting/abdominal pain), and neurologic dysfunction (confusion/weakness). Acute cardiac toxicity has broad EKG manifestations, but typically includes bradycardia, T-wave flattening/inversions, shortening of the QT interval, lateral ST depressions, and scooped ST segments. Bidirectional ventricular tachycardia while not pathognomonic should be considered digoxin toxicity until proven otherwise.

Multiple factors can exacerbate serum digoxin concentrations leading to digoxin toxicity. Renal insufficiency leads to poor digoxin clearance. Hypovolemia concentrates serum digoxin levels. Additionally, hypomagnesaemia, hypokalemia, and hypercalcemia sensitize the myocardium to digoxin's effects. Hypomagnesemia can increase myocardial



uptake of digoxin—so remember, it is vital to ensure serum magnesium levels are optimized in digoxin overdose!

The diagnosis of digoxin toxicity is not based on serum levels of digoxin; it is based on clinical presentation, ECG, and history. Serum digoxin levels do not always correlate with clinical toxicity, but can be used as an overall determinant for antidote dosing. In acute digoxin ingestion, serum digoxin levels should be measured upon arrival to the ED and again 6 hours post ingestion (in order for the serum levels to equilibrate). Patients admitted to the ED within 1 to 2 hours of an acute ingestion should receive activated charcoal. However, the definitive therapy for digoxin toxicity is digoxin immune Fab.

Digoxin immune Fab should be given to any patient with digoxin toxicity who have:

- Unstable cardiac arrhythmias
- Hyperkalemia
- Hypoperfusion leading to end-organ damage
- Acute ingestion >10 mg in an adult and >4 mg in a child

In acute digoxin toxicity, hyperkalemia is common. As ED physicians, our initial urge is often to treat this with calcium gluconate, insulin, glucose, and inhaled beta-2 agonists. However, with hyperkalemia secondary to digoxin toxicity, the preferred treatment is digoxin immune Fab, which restores the Na/K ATPase pump and drives potassium into the cells. Aggressive treatment of hyperkalemia with further agents like insulin/glucose in combination with digoxin immune Fab can lead to large potassium shifts and significant hypokalemia. In patients with digoxin toxicity and underlying hypokalemia (as is often seen in chronic toxicity), it is important to replete both potassium and magnesium when administering digoxin immune Fab in order to avoid further hypokalemia.

Digoxin immune Fab binds to digoxin to form inactive complexes that are unable to bind to digoxin's cellular sites of action. Many hospitals' serum digoxin levels will measure both free digoxin and the inactive complex bound to digoxin immune Fab, so remember after digoxin immune Fab has been given, the serum levels of digoxin no longer provide an accurate level of active digoxin in the body and cannot be used to guide further treatment.

Digoxin's narrow therapeutic window leaves patients susceptible to both acute and chronic toxicity. As an ED physician, it is important to pay special attention to serum electrolyte levels when treating digoxin toxicity. Hypomagnesaemia leads to increased cardiac uptake of digoxin, and hypokalemia can quickly occur when administering digoxin immune Fab.

Additionally, remember to avoid following serum digoxin levels after administration of digoxin immune Fab as they will often be inaccurate. When in doubt, you always have your regional poison control center available for urgent clinical consults!

## KEY POINTS

- The therapeutic window for digoxin is very narrow, leading to both acute and chronic digoxin toxicity. Renal insufficiency leads to reduced digoxin clearance and elevated serum digoxin levels.
- Digoxin serum levels will not always correlate with the clinical picture. Patients may be asymptomatic with elevated levels in an acute ingestion and will only become symptomatic when the drug has equilibrated into the cells.
- Hypomagnesaemia can increase myocardial uptake of digoxin. Ensure serum magnesium levels are optimized in digoxin overdose.
- After administration of digoxin immune Fab, serum concentrations of digoxin are no longer accurate. Use the patient's clinical picture to determine if further digoxin immune Fab is needed.
- Digoxin immune Fab restores the function of the Na/K ATPase pump which drives potassium intracellular, beware of rapid hypokalemia with treatment.

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## **DID YOU CONSIDER INTRAVENOUS LIPID EMULSION THERAPY?**

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Toxic ingestions are both a common and difficult emergency department presentation. Patients may have accidentally ingested medications, or there may be intentional ingestions as seen in suicide attempts. There are a variety of treatments for toxic ingestions, and it is important that we remember to approach each ingestion with these options in mind. Specifically, it is imperative that we remember to include intravenous lipid emulsion (ILE) in our treatment repertoire against the negative effects of toxins.

ILE therapy was first reported in 1962 to reverse the neurologic effects of barbiturate toxicity in rats. The first human case report was documented in 2006 in a case of cardiac arrest due to bupivacaine toxicity. Despite this history, there is a limit to both our understanding of the pharmacokinetics of ILE therapy as well as data regarding its use. While there are animal studies, human subject research is limited to case reports. There are two dominant theories regarding how ILE therapy works in the treatment of overdose. The first is known as the “lipid sink” theory, also known as partitioning theory, in which it is believed the lipid emulsion creates a concentration gradient, which attracts the lipophilic agent out of serum and therefore away from receptors due to the agents high lipid solubility. This approach has been shown to be incomplete by the successful reports involving more lipophobic drugs. The second theory focuses on the ability of phospholipids and triglycerides to be used as an alternate energy source for cardiac myocytes. As some local anesthetics demonstrate the ability to block the activity of carnitine-acylcarnitine translocase (CACT), they are able to prevent long-chain fatty acids from entering the cell’s mitochondria where they are needed

as an energy source. In this theory, it is believed that the large amounts of lipid are able to overcome this blockade and provide an energy source thereby reversing cardiac depression. A common critique of this model is its failure to explain why ILE also reverses noncardiac effects, such as neurologic toxicity.

Case reports have demonstrated successful use of ILE most commonly for local anesthetic systemic toxicity (LAST) due to agents such as bupivacaine and lidocaine; however, its use has also been demonstrated in a variety nonlocal anesthetic toxic ingestions such as amitriptyline, citalopram, bupropion, venlafaxine, quetiapine, verapamil, diltiazem, propranol, amlodipine, diphenhydramine, cocaine, and many more. Both cardiac and neurologic improvements are noted when ILE is administered to patients in these case reports. Surprisingly, success has been demonstrated in nonlipophilic ingestions, such as metoprolol and lamotrigine. While there is no consensus on the preferred formulation, the exact dosing regimen, or indications for use, there are some recommendations available. The current American Society of Regional Anesthesia and Pain Medicine (ASRA) recommendation endorses the use of ILE for the treatment of LAST and recommends a bolus of 20% lipid emulsion therapy at 1.5 mL/kg IV over 1 minute with a repeat bolus as needed for persistent cardiovascular collapse, followed by a continuous infusion of 0.25 mL/kg/min for at least 10 minutes after hemodynamic recovery. They recommend an upper limit of 10 mL/kg within the first 30 minutes. Despite this guidance, there is a wide variation among clinical practice, and use is recommended in consultation with Poison Control. There have also been documented cases of good outcomes both in pediatric cases as well as in pregnancy.

Despite many positive outcomes, there have been some documented side effects, which are believed to be associated with the use of ILE therapy. Commonly documented side effects include analytical failure due to the high lipid levels in blood samples after administration, which affect the ability to correctly interpret some laboratory values, hyperlipidemia, pancreatitis, and acute respiratory distress syndrome, which, some reports have shown, may be less likely with decreased infusion rates.

As physicians, we often play an important role in the diagnosis and initial treatment of toxic ingestions. It is essential that we are thoroughly prepared for this task and are aware of treatment options at our disposal. When dealing with toxic ingestions, it is important to first and foremost remember to stabilize the patient using our very familiar ABCs. Subsequently, as we continue to manage a patient who is ill due to toxic ingestion, we should be mindful of our resources including conferring with poison control, being prepared to treat the ingestions with medications

available to us and considering ILE therapy as an optional course of action.

## KEY POINTS

- While classically considered for LAST with agents such as bupivacaine, many other agents have had successful response to ILE.
- ILE is commonly dosed according to the ASRA guidelines as a bolus of 20% lipid emulsion therapy at 1.5 mL/kg IV over 1 minute. A repeat bolus may be given if there are no signs of clinical improvement. This should be followed by a continuous infusion of 0.25 mL/kg/min for at least 10 minutes after hemodynamic recovery.
- While there are noted treatment-associated side effects such as pancreatitis, ARDS, and hyperlipidemia, many of these are related to the duration of infusion. Of note, when giving ILE, one should expect laboratory value derangement associated with giving intravenous lipid.

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# MANAGING THE HOT AND BOTHERED: SYMPATHOMIMETIC OVERDOSES

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**ARUN NAIR, MD, MPH**

In emergency medicine, sooner or later, this challenging patient will present: you're unable to get a good history, their vital signs are all out of whack, they're impossible to keep in bed or start an IV line on, and your nurses are completely frustrated. Here's how to keep everyone calm:

Treat the patient first and then numbers

Remember the sympathomimetic toxidrome: hyperthermic, tachycardic, hypertensive, and diaphoretic—so what to treat first? Patients with sympathomimetic toxicity are in a hyperadrenergic state due to whatever it is they took. Regardless of the particular substance they ingested, agitation is exacerbating their vital sign instability. If you want take care of the patient, relieve their agitation first. Don't waste time trying to figure out which exact drug he or she took as it doesn't matter in most cases. Like many other ingestions without a particular reversal agent (and even some that have one), your goals are symptom control, patient safety, and allowing for metabolism and/or clearance to take care of the rest. The nuances of each possible ingestion in this class are not something that needs to be memorized or agonized over to deliver excellent care. Too often, patients present with polysubstance ingestions making the diagnosis of a particular drug's toxidrome even more difficult. Sure, get the urine toxicology screen, but there are many derivatives these days that won't show positive. Just focus on the presenting symptoms and treat those. Your patient is agitated and needs sedation first. What is the first-line agent in this case?

# **BENZOS, BENZOS, BENZOS**

Benzodiazepines are the answer—in liberal quantities until effect is reached. There's a lot of hemming and hawing on which drug is best, but the real answer is whichever drug you and your staff are most familiar with and comfortable using. That being said, you should consider starting with diazepam (Valium) given its quick peak of onset and short duration of effect, despite its known active metabolites. Repeat dosing of diazepam every 5 to 7 min generally does the trick (5, 10, 10, 20 mg); the only mistake is being too timid in dosing and failing to achieve adequate sedation, or not allowing for enough time between dosings. Transition to IV dosing as soon as possible as IM effects are less predictable. Yes, these medicines are respiratory depressants, but dosing in a calm and methodical manner will almost never cause a truly agitated patient to lose their airway. These patients tend to be younger in general, which also allows them to tolerate the benzodiazepines quite well. Though Precedex is on the horizon after having proven itself in the ICU setting, most of us don't have access to this in our emergency departments, our staffs are not familiar with it, and it requires continuous infusion after an initial load. Antipsychotics have too long of a time of onset (15 to 20 minutes even for IV), last 6 to 8 hours, and can lower the seizure threshold a patient with unknown medical history. Some, however, promote its use as an adjunct, giving a single dose with the initial round of benzodiazepine. If intubation does become necessary, avoid ketamine (disassociating an agitated patient may not be the best plan) and avoid depolarizing agents, which prolong cocaine's effects and could worsen hyperkalemia or could potentiate arrhythmias in rhabdomyolysis/hyperthermia.

## **CARDIOVASCULAR EFFECTS**

Once your patient is adequately sedated, reassess vital signs. If the patient remains significantly hypertensive (DBP > 100), further intervention may be required as sympathomimetics exert their vasoactive effects through both alpha-1 and beta activation. Any focal neurologic symptoms including severe headaches necessitate a head CT, and chest pain always begets a repeat EKG. (These patients all require an initial EKGs). In severe overdoses, cocaine can produce sodium channel blockade with resultant QRS widening and negative inotropy. If QRS widening occurs, bolus amps of  $\text{NaHCO}_3$  until the complex narrows. Be aware of the increased risks from concurrent use of cocaine and alcohol. The resultant metabolite cocaethylene potentiates the neurotoxic and cardiotoxic effects of either drug alone.

Though the underlying evidence has been called into question, it remains standard practice to avoid the use of beta-blockers as antihypertensive due to the resultant unopposed alpha stimulation. Here the drugs of choice are phentolamine (5 to 10 mg IV q10min) or a nitroprusside drip (start at 0.3 mcg/kg/min, titrate by 0.5 mcg, max 10 mcg/kg/min). If you are starting one of these vasoactive medications, your patient needs an arterial line. Period.

## **HYPERTHERMIA**

Most patients' hyperthermia will resolve or significantly improve once adequately sedated. A caveat is that these ingestions (MDMA/designer/cocaine) often occur in hot club/dance settings where the patient has significantly over-exerted and dehydrated him or herself. This should raise your concern for the possibility of rhabdomyolysis, especially if there is ongoing myoclonus or tremors. Start the intravenous fluids, and monitor the creatine kinase levels. Though ice bath immersion is the most expedient method of dropping core temperature, it's easier said than done. If core temperature remains >102°F, fully expose the patient, pack the protected groin and axilla with ice, wet the skin (spray bottles work great), and get fans blowing directly on the patient to achieve "wet and windy." This is a very effective method for cooling, and the benzos on board should help prevent counterproductive shivering. Be sure to recheck core temperature frequently so that you don't overshoot to hypothermia. Placement of a temperature-sensing Foley in these patients (once adequately sedated) for continuous measurement of temperature and urine output is an excellent idea.

## **CONCOMITANT INJURY**

These patients are most often part of a vulnerable population at high risk for unmanaged medical comorbidities and traumatic injury that should not be overlooked while treating the toxidrome. Furthermore, your patients had to get the offending agent into them somehow, and these routes could be underlying problems. Smoking crack and amphetamines requires high temperatures, which can cause burns to the airway, pulmonary edema, pneumothorax, and crack lung. These patients often have underlying emphysematous changes, which could also be in acute exacerbation. Bleeding ulcers should be considered in cocaine users. If you practice in an area with significant drug trafficking, your patient may be a body stuffer, and whole bowel irrigation or surgical intervention may be required. And lastly, be sure to completely expose your patient and look for signs of skin infection at shooting sites as you could be missing developing sepsis as you trek your



way down the toxicology rabbit hole.

## KEY POINTS

- Once the toxidrome is identified, prioritize resolving patient agitation over substance identification or pharmacologic management of vitals abnormalities.
- Benzodiazepines are the drug of choice and should be administered in a methodical and not in a reactionary manner.
- Sympathomimetics are potent alpha and beta stimulators with cardio-/neurotoxic effects, and patients should be screened for vascular injury. Resultant hypertension may require reversal with agents other than beta-blockers.
- Agitation and the circumstances of the sympathomimetic ingestion increase the patient's risk for hyperthermia and rhabdomyolysis, which may require monitoring of temperature and urine output.
- Sympathomimetic overdoses often occur in vulnerable population with comorbidities and risk for concomitant injury that require investigation.

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## EMERGING DRUGS OF ABUSE

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**CHELSEA WILLIAMSON, MPAS, PA-C**

Influenced by cultural, socioeconomic, and legislative facets of different communities, the trends of substance abuse are constantly evolving. Over the past three decades, synthetically derived compounds have made the most profound impact among developed nations. This chapter will highlight some of the newest trends and discuss tips for recognizing and managing patients in the emergent setting.

### SYNTHETIC CANNABINOIDS

Commonly known street names: Spice, K2, Black Mamba, Cloud 9, Mad Hatter, Aztec Gold, and many more.

Delta-9-tetrahydrocannabinol (THC) is the naturally occurring active chemical in the marijuana plant. When ingested, THC will bind to cannabinoid receptor type 1 (CB<sub>1</sub>) receptors within the central nervous system, increase dopamine release, and cause effects such as euphoria, relaxation, heightened sensory perception, altered perception of time, increased appetite, anxiety, and paranoia. In the 1980s, researchers derived synthetic cannabinoids for the purpose of potential medical therapies. Compared to THC, a partial agonist with a relatively low affinity, these synthetically derived substances, for example, naphthoylindoles and cyclohexylphenols, are full agonists with affinities of up to 200 times that of THC and are associated with significant, and in some cases life-threatening, adverse effects. Many presenting symptoms are the same as those seen with marijuana use, but additional findings such as violent behavior, agitation, hallucinations, hypertension, hypokalemia, and seizures should increase clinical suspicion of synthetic cannabinoid ingestion. Complications of toxic ingestions include seizure, self-injury, myocardial infarction, preeclampsia in

pregnant women, acute renal failure, and coma.

## SYNTHETIC CATHINONES

Commonly known street names are Bath Salts, Ivory Wave, Flakka, Purple Wave, Red Dove, Blue Silk, Zoom, Bloom, Cloud Nine, Ocean Snow, Lunar Wave, Vanilla Sky, White Lightning, Scarface, and Hurricane Charlie.

Cathinone is the active ingredient naturally found in *Catha edulis* (commonly known as khat), a plant found in the Horn of Africa and the Arabian Peninsula. Traditionally extracted by chewing leaves and twigs of the shrub, cathinone is structurally similar to amphetamine, which acts in the brain as a stimulant by targeting monoamine transporters and increasing dopamine, serotonin, and norepinephrine concentration in the synaptic cleft between neurons. In lower doses, cathinone is known for effects such as increased alertness and euphoria. Beginning in 2010 in the United States, laboratory derivatives of cathinones, commonly known as bath salts, have been synthesized for recreational use. Methylenedioxypyrovalerone (MDPV), 4-methylmethcathinone (4-MMC), mephedrone, flephedrone, or methylone are among the most commonly synthesized compounds, which are 10 to 50 times more potent than the cathinone found in khat. These synthetic cathinones are sold in powder form and can be ingested orally, inhaled, injected, and insufflated. Common clinical effects include those of the sympathomimetic symptoms (tachycardia, mydriasis, diaphoresis, hypertension, and hyperthermia), as well as dopamine- and serotonin-related psychosis, hallucinations and agitation. In the most severe cases, hyperthermia, seizures, and intracranial hemorrhage may occur.

## PIPERAZINE DERIVATIVES

Commonly known street names are Party Pills, Legal Ecstasy, Benzo Fury, MDAI, Head Rush, Exotic Super Strong, and XXX Strong as Hell.

Originally synthesized in the laboratory as antihelminthic agents, the active compound N-benzylpiperazine (BZP) was found to have amphetamine-like effects by stimulating serotonin release as well as inhibiting its reuptake. Since 2004, the use of the piperazine derivatives in recreational drug use has exponentially increased along with simultaneous development of newer generations of compounds similar to BZP. Effects will last between 6 to 8 hours after use. Symptoms include euphoria, hyperactivity, increased energy, headache, vomiting, anxiety, tachycardia, prolonged QT, and seizure.

As with most emerging drugs of abuse, little has been studied regarding the management of these specific ingestions. As such, there are unfortunately no antidotes, and patients must be symptomatically treated on a case-by-case basis. If a patient is unable or unwilling to provide a history of synthetic cannabinoid, cathinone, or piperazine use, then diagnosis may be challenging. As these compounds are undetectable on routine toxicology screens, providers must base their management on history and clinical presentation alone. Due to the hostile and violent nature of patients who have ingested these synthetic compounds, intramuscular sedative cocktails are commonly administered to maintain a safe environment for both the patient and staff members. If there is any suspicion of synthetic drug use, first-generation antipsychotics such as haloperidol should not be given, as they may lower the patient's seizure threshold. Benzodiazepines in repeat doses and physical restraints are the recommended interventions for control of the aggressive patient. Associated conditions such as dehydration, hyperthermia, and electrolyte disturbances should be managed using supportive care measures.

As quickly as regulations evolve to restrict the sale of these harmful substances, so do the manufacturing processes in which they are produced. The timely changes to the chemical framework of these synthetic compounds allow for their legal sale under the pretense that they are “not for human consumption.” As they are sold legally and they are easily accessible, their popularity as recreational drugs within the United States and Europe continues to grow, as do their potential complications.

## KEY POINTS

- Synthetic compounds are undetectable on routine toxicology screens.
- Medications known for lowering seizure threshold, such as haloperidol, should not be given to patients with suspected synthetic compound ingestion.
- Benzodiazepines and other supportive care measures are the mainstay treatment for aggressive patients, as there is no antidote to treat ingestion of synthetic compounds.
- Synthetic cannabinoids have been known to cause symptoms for up to 48 hours or longer after ingestion.
- The popularity of synthetic compounds use is growing at an exponential rate and should be high on the differential for patients presenting with unexplained aggression and altered mental status.

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## CYANIDE POISONING: A TALE OF TWO ANTIDOTES

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Cyanide poisoning is a rare occurrence in the emergency department, but the rapid onset of hemodynamic instability and unique antidotal therapy makes it a diagnosis that an emergency physician cannot overlook. In 2007, the American Association of Poison Centers reported five deaths from cyanide, which is as many as from digoxin and beta-blockers. The most common cause of cyanide toxicity comes from smoke inhalation during house and industrial fires with recent evidence suggesting that many of the immediate deaths are caused by inhaled cyanide.

Cyanide's toxicity derives from its ability to react with high affinity with metals such as ferric iron and cobalt allowing it to bind to numerous critical enzymes in the body. Most notably, it competitively inhibits cytochrome c oxidase (Complex IV) the final enzyme in the electron transport chain causing cessation of oxidative phosphorylation. This ultimately leads to histotoxic hypoxia, the inability of cells to utilize oxygen from the bloodstream despite normal delivery.

The early clinical manifestations of cyanide toxicity stem from the histotoxic hypoxia and the body's attempt to increase oxygenation through tachycardia and tachypnea. These early symptoms can rapidly progress to profound hypotension, cardiac arrhythmias, and cardiac arrest. Shock and cardiac arrest can occur in about 50% of cyanide-exposed patients. Additionally, laboratory investigations reveal elevated plasma lactate concentrations often greater than 10 mmol/L.

In the United States, there are two therapies approved by the Food and Drug Administration for the treatment of cyanide toxicity: hydroxocobalamin

and sodium nitrate. Essentially, these antidotes work by shunting the cyanide into a nontoxic form. Both antidotes are then supplemented with sodium thiosulfate, which acts as a sulfhydryl donor in the enzymatic conversion of cyanide to the relatively nontoxic thiocyanate.

Hydroxocobalamin works by chelating cyanide to form the nontoxic metabolite cyanocobalamin (vitamin B<sub>12</sub>). This water-soluble vitamin is then excreted in the urine. The administration of hydroxocobalamin is associated with transient hypertension as well as temporary reddening of the skin and urine. Given hydroxocobalamin's red color, it can interfere with certain colorimetric laboratory tests, such as creatinine, carboxyhemoglobin, methemoglobin, and oxyhemoglobin.

Sodium nitrite's mechanism of action derives from its ability to oxidize the iron in hemoglobin and cause a methemoglobinemia. The methemoglobin then binds the circulating cyanide and provides the body the opportunity to detoxify the poison. Given that the methemoglobinemia reduces the level of functional hemoglobin, it may be harmful to patients who already have a deficiency in oxygen carrying capacity such as those exposed to carbon monoxide or anemic patients. Additionally, the use of sodium nitrite can lead to hypotension given the potent vasodilatory action of nitrites.

To date, there have been no human randomized trials comparing the efficacy of the two antidote combinations. Recently, a swine model comparing the two antidote combinations showed no difference in mortality.

Overall, the most important step in treating cyanide toxicity is early recognition. Once identified, it is important to know which antidotes are readily available in the local emergency department for treatment.

## KEY POINTS

- The most common cause of cyanide toxicity comes from home and industrial fires.
- Cyanide prevents cells from utilizing oxygen resulting in a profound plasma lactate elevation.
- Sodium nitrate and hydroxocobalamin are two approved treatments for cyanide toxicity.
- Hydroxocobalamin interferes with colorimetric laboratory tests.
- Swine studies show no difference in mortality between hydroxocobalamin and sodium nitrate.

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# METHEMOGLOBINEMIA: BLUE PEARLS

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## WHAT IS IT?

Methemoglobinemia is one of several *dyshemoglobinemias* resulting in a *functional anemia* where the heme group is oxidized from 2+ to 3+ making it incapable of delivering oxygen. The oxygen dissociation curve of the remaining *functional* hemoglobin is shifted left resulting in *additional* impairment of oxygen delivery. Symptoms are essentially what you would expect with an anemic patient reflecting a shock state at high levels.

## HOW TO NOT MISS IT

Methemoglobinemia is rare, and etiologies can be difficult to commit to memory. Thankfully, *most* patients with a significant methemoglobinemia will be *blue in the face* trying to tell you. It takes 1.5 g/dL of methemoglobinemia to exhibit cyanosis, compared to about 5 g/dL of desaturated hemoglobin, making cyanosis *relatively* sensitive. Cyanosis due to hypoxia should be associated with prominent pulmonary findings (or history), as 5 g/dL of desaturated hemoglobin would correspond to an O<sub>2</sub> saturation of 67% in a patient with a hemoglobin level of 15!

If you miss the diagnosis in an *acyanotic* patient, chances are the diagnosis will be clinically inconsequential in healthy patients. A drop in *functional* hemoglobin from 15 to 14 g/dL is relatively benign—even with the leftward shift of the dissociation curve. However, if a patient has a

*preexisting* anemia, a significant methemoglobinemia could exist without the blue courtesy warning (cyanosis). A patient with an Hgb of 7.0 g/dL and with 1 g/dL of methemoglobin may *not* be cyanotic but could easily be symptomatic. In these cases, it may help to be familiar with those most common causes.

## WHAT CAUSES IT?

Extensive tables of causes can be found, but this is rarely clinically useful, and impractical for memorization. Many *environmental* exposures can cause methemoglobinemia making the etiology difficult to identify. Though individuals with hereditary methemoglobinemia will likely be aware of their condition, they may not be able to communicate while in extremis. Knowing the cause(s) is probably *less* important for diagnosis and *more* important to avoid iatrogenic methemoglobinemia in at-risk patients with a preexisting anemia or comorbidities. See [Table 254.1](#) for a list of high-yield causes.

**TABLE 254.1 HIGH-YIELD CAUSES OF METHEMOGLOBINEMIA**

| CLINICAL PROMPT    | DRUG                         | NOTES  |
|--------------------|------------------------------|--|
| Oral anesthesia    | Hurricane spray (benzocaine) | Even low doses   |
| UTI                | Pyridium                     | Available OTC and Rx                                   |
| Third world travel | Antimalarials                | “Quine” drugs  |
| HIV                | Dapsone                      | Alternative PCP prophylaxis                            |
| Cyanide toxicity   | Amyl and sodium nitrite      | Used clinically in cyanide poisoning, occasional abuse |
| Drug Abuse         |                              |  |

## HOW TO DIAGNOSE IT

Blood with methemoglobin classically has a chocolate brown color. Many sources state that the pulse oximeter will read 80% to 85%, though this may be unreliable with levels <30%. An O<sub>2</sub> saturation of 80% to 85% may prompt investigation in the correct context, but a different value should not be used to rule out methemoglobinemia.

An ABG and bedside pulse oximetry should be ordered for initial diagnosis. Methemoglobin can cause a false elevation of the oxygen saturation on an ABG if your lab reports calculated saturations from the PaO<sub>2</sub>. This would not correspond with the oxygen saturation seen on pulse oximetry and result in a “saturation gap.” For definitive diagnosis, venous or

arterial **co-oximetry** should be performed.

## HOW (AND WHEN) TO TREAT

A 1.5-g level of methemoglobin corresponds to 10% in a patient with 15 g of hemoglobin and will be benign in an otherwise healthy individual. You can be blue and happy! In this population, simply removing the offending agent (if identified) can be adequate. Activated charcoal and other methods of decontamination are likely to be of limited value, but should be considered in the case of proximate ingestions or exposure to a likely cause.

The general rule of thumb is to **treat when methemoglobin levels are significant *and* the patient is symptomatic or when methemoglobin levels are >25% even if asymptomatic.** A specific threshold for treatment cannot be given as this will vary based on preexisting anemia and comorbidities, but keep in mind that healthy people can have baseline methemoglobin levels of up to 2%.

Treatment consists of **1 to 2 mg/kg methylene blue over 5 to 15 minutes.** Infusion should be slow, as rapid administration can *induce* methemoglobin formation. Clinical improvement should be seen within an hour, and repeat administration and/or transfusion should be considered at this time. Benefits must be balanced with the risks of a G6PD deficiency. Most patients will not know if they have this, and it is impractical to screen patients prior. Treatment is an **exchange transfusion** if G6PD is known. If the patient is severely symptomatic and the resources or time do not allow for an exchange transfusion, **standard PRBC transfusion may be appropriate.** Special consideration should be given in pediatrics. Infants have underdeveloped reduction mechanisms, making them more susceptible to methemoglobinemia, particularly from environmental factors. To make matters worse, some sources list the use of methylene blue as “not recommended” under the age of 6, though Lexicomp states “refer to adult dosing” for pediatric dosing. Clinical judgment should be used.

### KEY POINTS

- Methemoglobinemia decreases oxygen delivery by *two* mechanisms.
- Don’t spend too much energy looking for a cause; many are obscure or environmental.
- You can be blue and happy. Not all cyanotic patients need to be treated, but don’t let the fear of G6PD deficiency stop you from

treating a patient in shock!

- Think of methylene blue for methemoglobinemia like Narcan in a methadone overdose in that the half-life of methylene blue is short—but that of the drugs causing it can be long.
- Avoid prescribing drugs that can cause methemoglobinemia in patients who have preexisting anemia.

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## SHOULD I TAKE THAT?

### NUTRITIONAL SUPPLEMENTS

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**EDMUND TIMPANO, MD AND SARIKA WALIA, MBBS**

Nutritional supplements are used by a majority of the US population.

Multivitamins are among the most commonly used dietary adjuncts. Vitamins are organic compounds essential for basic metabolism that cannot be intrinsically produced in sufficient quantities or at all. They must be obtained from extrinsic dietary sources, or else a deficiency can develop. Vitamins can be classified as being either water soluble or fat soluble. Healthy adults and nonpregnant women can obtain all the necessary vitamins required from a balanced diet, and the American Medical Association does not recommend for their routine daily use. If supplemented inappropriately, some of these vitamins can have serious potential toxicities requiring emergent medical attention.

The majority of vitamins are water soluble. These include thiamine (B1), riboflavin (B2), niacin (B3), pantothenic acid (B5), pyridoxine (B6), biotin (B7), folic acid (B9), cyanocobalamin (B12), and ascorbic acid or vitamin C. Water-soluble vitamins are readily excreted instead of being stored and therefore have limited toxicity in excess. Of these, only niacin, pyridoxine, and vitamin C have potential toxicity.

Niacin, or nicotinic acid, a vitamin popularized for its use for hyperlipidemia, has well-known reactions when taken in excess. Normal dietary consumption of niacin is typically benign. Doses of nearly 100-fold the recommended daily allowance are used for hyperlipidemia treatment and can cause symptoms. Acutely, skin flushing, redness, and pruritus secondary to its vasodilatory properties are most common. Premedication with aspirin or antihistamines can help. More prolonged effects of excessive niacin intake

may include elevated glucose levels, skin hyperpigmentation, hyperuricemia, liver dysfunction, and hepatotoxicity. Deficiency is relatively rare but can cause pellagra, which is characterized by the triad of diarrhea, dermatitis, and dementia.

Pyridoxine, vitamin B6, is known to be toxic in excess. Interestingly, both toxicity and deficiency of this vitamin can have deleterious effects on the nervous system. Deficiencies can cause both a peripheral sensory neuropathy as well as seizures. This is because pyridoxine is a precursor to the inhibitory neurotransmitter, GABA. Medications tend to be the leading cause of deficiency, most notably isoniazid. The definitive treatment of seizures in such cases is restoring pyridoxine stores. When taken in excess, patients can develop more subacute and chronic peripheral neuropathies. Patients may have loss of sensation in the feet and hands and impaired proprioception that is potentially reversible with termination of supplementation.

Vitamin C is found in most fruits and vegetables and helps to facilitate iron absorption in the body as well as aids in the synthesis of collagen. Deficiencies in vitamin C cause scurvy with weakened vessels and easy bleeding. Once thought to cause nephrolithiasis in excess, there has actually been no definitive studies linking the two. More consistently, hypervitaminosis C can cause osmotic diarrhea, gouty flares, and esophagitis with extended contact.

Fat-soluble vitamins—D, E, A, and K—unlike water-soluble vitamins, are often stored in large amounts. Consequently, they will amass in excess with greater toxicity potential. With the exception of potential anaphylaxis when administering vitamin K intravenously, excess vitamin K has limited toxicity.

Vitamin E is an essential nutrient believed to be important in numerous body systems and for antioxidant effects. The nervous system seems to be primarily affected when lacking. There can be peripheral neuropathies and poor position and vibration sensation leading to trouble walking and imbalance. In toxicity, gastrointestinal symptoms are common along with fatigue, visual trouble, and also thrombophlebitis. Active vitamin K levels may also be reduced, but coagulopathy is rare unless in presence of other vitamin K antagonists.

Vitamin A, or the retinoids, play important roles in many bodily functions. Retinol is the main bodily storage form in the liver. It is known for its beneficial skin effects and, in other forms, for its critical role in normal vision function and retinal health. Deficiency affects the normal epithelium leading to skin changes, excessive keratinization of the cornea

(xerophthalmia), and trouble seeing in dim lighting. Too much vitamin A has concerning consequences as well. Retinoids, commonly used for acne treatment, must be avoided in pregnancy as they are known teratogens. Consumption in excess has also been linked to idiopathic intracranial hypertension also known as pseudotumor cerebri with headaches and papilledema. In acute ingestions, hepatotoxicity can develop in hours to a few days. Classically, there can be gross yellow-orange skin discoloration that spares the sclera. More prolonged intake of excess vitamin A can cause liver fibrosis and even cirrhosis. Especially in areas heavy in animal liver consumption, persons can develop a progressive gait imbalance with associated mood fluctuations and even florid psychosis.

Vitamin D functions in calcium homeostasis by increasing calcium absorption and stimulating bone turnover and calcium release. Ergocalciferol, vitamin D<sub>2</sub>, and cholecalciferol, vitamin D<sub>3</sub>, are the two forms found in humans. Both are biologically inactive and must be metabolized to the active form, calcitriol. When lacking, the bones are the most commonly affected system leading to the development of rickets in children and osteomalacia in adults. The most concerning effect from hypervitaminosis D is hypercalcemia. Therefore, the manifestations of excess vitamin D tend to mimic those of hypercalcemia with weakness, fatigue, and dehydration early. Later, bone pains can develop with constipation, renal insufficiency, and even psychosis. Treatment is aimed at correcting the hypercalcemia and removing all supplemental vitamin D and calcium from the diet. Additionally, steroids may be considered as they are believed to help prevent further calcium absorption from the intestines.

Emergency department care for hypervitaminosis is typically supportive care only. The offending agent should be stopped. Gastric decontamination can be considered for known, acute vitamin A or D overdoses with activated charcoal. Hypercalcemia due to vitamin D toxicity should be managed with standard care including fluids and furosemide. Steroids can be considered, but data are limited. Pseudotumor cerebri, especially with papilledema and visual changes, in the setting of excess vitamin A may require a diagnostic and therapeutic lumbar puncture. N-acetylcysteine can be considered in the setting of acute hepatotoxicity from suspected hypervitaminosis A.

## KEY POINTS

- Routine vitamin supplementation is not needed in otherwise healthy persons and nonpregnant women with a balanced diet.

- Water-soluble vitamins—the B vitamins and vitamin C—are readily excreted and tend to have limited toxicity in excess.
- In most circumstances, acute hypervitaminosis is benign; greater toxicity is more common with prolonged supraphysiologic vitamin supplementation.
- Treatment of hypervitaminosis is generally supportive and withdrawal of the offending agent.
- Management complications of hypervitaminosis complications, for example, hypercalcemia and pseudotumor, should follow their standard therapeutic practices.

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## **SECTION XIX**

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# **TRAUMA/ORTHO**

## ELECTRICAL INJURIES: SHOCKING OR SUBTLE?

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**TIM HORECZKO, MD, MSCR, FACEP, FAAP**

Electrical injuries present in two forms: (1) cardiovascular instability (rarely) or, more commonly, (2) well with subtle symptoms and signs. In unstable patients, treat the presenting dysrhythmia (most often ventricular fibrillation or asystole) and protect against hypoxia-induced secondary dysrhythmias. In the well-appearing patient, be mindful of potential missteps—the initial complaints can be vague and may include ill-defined pain, anxiety, or weakness. *Here are some key points to consider on history that might help identify potential injuries.*

### **WAS IT HOUSEHOLD OR INDUSTRIAL?**

Household electricity uses alternating current (AC), 100 to 240 volts (V), and may cause dysrhythmias and burns. Industrial electricity (and lightning) is delivered as direct current (DC). Contact with high voltage (AC > 1,000 V; DC > 1,500 V) can repel a victim and cause subsequent trauma, or it may trigger tetany and resultant longer contact with the high-voltage current. Patients with DC injuries may have injuries not apparent initially: deeper structures such as bone, tendon, and fat have a higher resistance to current than do skin and mucous membranes. Increased resistance to electrical current will heat deeper tissues, causing coagulation and necrosis not readily visible externally; thus, *serial examinations and observation are necessary in high-voltage injuries.*

### **WHAT PATHWAY DID THE CURRENT TAKE?**

Did the current pass through the thorax? (*Think dysrhythmias.*) Through the head or neck? (*Think central respiratory arrest, acoustic nerve damage, or cataracts.*) Along an extremity? (*Think compartment syndrome and rhabdomyolysis.*)

## WHAT WAS THE CONTACT TIME?

Electrical charge converts to thermal energy and causes tissue necrosis with significant contact time, typically considered any duration longer than immediate release or repulsion. The longer the contact time, the more electrical charge is converted to thermal energy with subsequent tissue necrosis. Patients with a history of tetany or need for extrication are at highest risk for extensive thermal injury with delayed symptoms, which can present hours after the injury.

## ARE THERE ASSOCIATED INJURIES?

Was the patient's body flung after contact (*associated trauma*)? Did he have a syncopal episode (*presumed dysrhythmia*) or chest pain (*stress-induced ischemia*)? Patients may be confused initially or unable to localize symptoms due to CNS disruption. Get collateral information, reinterview, and reexamine as needed.

### ***A Prototypical Pitfall in an Adult***

A 42-year-old man stumbled while cleaning the gutters on his roof and grasped a semiexposed power line briefly before falling from his feet to his buttocks onto the roof itself. No trauma was sustained, and he presents to the ED asymptomatic. His vital signs and examination are normal. An ECG shows mild right ventricular conduction delay (none available for comparison), and he was sent home.

Hours later, he returns with dizziness, arm and shoulder pain, and is in sinus tachycardia with runs of nonsustained ventricular tachycardia, early forearm compartment syndrome, rhabdomyolysis, and acute kidney injury.

### ***Lesson***

Although most outlets in the United States use 110 V, power lines to a residence supply 220 V for large appliances—this man received a much higher voltage than assumed. The abnormal ECG (i.e., right bundle-branch block) is an electrical injury until proven otherwise. And with one organ

system affected (cardiovascular), others should be investigated.

## ***An ECG Is Recommended in All Patients***

Other screening tests may include a chemistry panel, creatine kinase, and urinalysis, as directed by the history and physical examination. The diagnostic or prognostic utility of troponin in electrical injury is not clearly defined.

## ***A Prototypical Pitfall in a Child***

An 18-month-old boy sustained an oral commissure burn (e.g., at angle of the mouth) after chewing on an old extension cord. A hemostatic second-degree burn was present on the right oral commissure. He was otherwise well appearing, successfully took fluids, and was therefore discharged home.

One week later, he arrives in hypovolemic shock after significant blood loss from a delayed labial artery injury. He is resuscitated, intubated, and requires emergent surgery.

## ***Lesson***

This toddler was at risk for significant delayed bleeding. The electrical burn cauterizes superficial bleeding vessels, and hours later, the wound is covered with a white layer of fibrin, surrounded by erythema. Edema and thrombosis continue, and at 24 hours, there is typically a significant margin of tissue necrosis. When the eschar sloughs off in 1 to 2 weeks, the labial artery is exposed, leading to a potentially life-threatening bleed. Parents should be warned of this possibility and be prepared to render basic first aid.

Show parents how to apply pressure to either (superior or inferior) labial artery: *“If the wound bleeds, pinch the outside cheek and the inside of his mouth between your thumb and finger, just deeper in the mouth to the bleeding. Hold constant pressure until you get to the hospital.”*

For adults and children, document thorough cardiovascular, neurologic, musculoskeletal, and skin examinations. The extent of injuries may evolve. Take pictures when feasible.

## **KEY POINTS**

- Injury from electrical burns can be subtle. Think of patients as having occult multitrauma. Be thorough in history and examination. Plan to

reexamine either during a period of observation in the ED or as part of close outpatient follow-up.

- Discharge patients only if the injury was of low voltage, if there is an absence of symptoms, and if the ECG is normal. Counsel outpatients, and provide close follow-up as appropriate.
- Admit patients with high-voltage injury and those with low-voltage injury and signs or symptoms (loss of consciousness, ECG changes, evidence of end-organ damage on laboratory screen).
- Transfer patients with high-voltage injury and/or significant burns to a regional burns or trauma center.

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# **“DON’T TASE ME BRO!” THE TASERED PATIENT IN THE ED**

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**PETER MILANO, MD, MHA**

The TASER (an acronym for Thomas A. Swift Electric Rifle) is the most widely used of the so-called conductive energy weapons or electronic control devices. The use of these devices by law enforcement has become commonplace, and patients are often brought to the emergency department (ED) for medical clearance after their use. The clinician must be able to confidently and quickly clear low-risk patients and, most importantly, appropriately further evaluate patients at higher risk for complications.

## **WHAT EXACTLY HAPPENS WHEN A TASER IS USED?**

In “drive stun” or “touch” mode, the device electrodes are pressed directly against the victim to deliver a painful shock. In “projectile” or “probe” mode, barbed probe electrodes are shot at the victim at a speed of about 50 m/s (164 feet/s), with a range up to 10 m (33 feet). The barbs embed into the victim’s skin or clothing and are tethered to the handheld device by wires. The barbs can cross up to 2 inches of clothing and deliver electricity in short pulses, leading to involuntary muscle tetany and pain. The typical duration of a shock is 5 seconds.

**POTENTIAL PITFALL #1: FAILURE TO CONSIDER PREDISPOSING/ASSOCIATED**

## **CONDITIONS TRIGGERING THE TASER ENCOUNTER**

Patients often have coexisting conditions such as psychostimulant use, alcohol intoxication, and excited delirium, which may have prompted the use of the TASER. In excited delirium, patients exhibit insensitivity to pain, diaphoresis, severe agitation, and unusual strength. These patients are at high risk for serious complications such as hyperthermia, rhabdomyolysis, metabolic acidosis, and sudden death. Physical restraint (with attention to protection of the airway and cervical spine) and aggressive chemical sedation are frequently required.

## **POTENTIAL PITFALL #2: FAILURE TO CONSIDER UNDERLYING SERIOUS INJURY**

Most injuries sustained from these devices are minor but severe injuries can occur. Patients are incapacitated by the shock and may fall without protective reflexes. Blunt trauma is often sustained and may result in injuries such as fractures and intracranial hemorrhage. Severe multisystem injury may be incurred after a fall from a height. Drowning may occur. Forceful muscle contraction related to the shocks has been implicated in scapula and spinal compression fractures. Penetrating injuries may occur from the impact of the barbs (globe rupture, lacrimal duct laceration, tracheal perforation, complex finger injuries, and penetrating intracranial injuries through the skull have been reported). Pneumothorax is possible in thin patients. Superficial skin burns may occur. Consideration of injury to underlying structures is particularly important with barbs embedded in vulnerable regions such as the head, face, neck, genitals, joints, and hands.

## **EMERGENCY DEPARTMENT MANAGEMENT OF THE TASERED PATIENT**

The history and physical examination dictates management of these patients in the ED. Laboratory studies, radiographic studies, and electrocardiograms are not indicated in asymptomatic patients without either prolonged exposure (>15 seconds) or features concerning for traumatic injuries or coexisting medical conditions. Routine ED observation or admission is not necessary. An electrocardiogram should be obtained in symptomatic patients (e.g., chest pain, shortness of breath, or palpitations) to evaluate for conduction

abnormalities or cardiac injury. Miscarriage has been reported with TASER use in a pregnant woman but a causal relationship is unclear. Observation may be indicated in pregnant patients when the fetus is viable. Pacemakers and implantable cardioverter defibrillators (ICDs) are susceptible to malfunction, and interrogation should be considered.

## HOW TO REMOVE THE EMBEDDED TASER BARBS

The barbs should be removed if simply embedded into soft tissue (and not in high-risk locations such as the eye). The barbed end of the probe is 9 mm long and has a barb blade on the end (like a straightened out fish hook attached to a larger cylindrical shaft). The direction of the blade of the barb beneath the tissue is aligned with the groove in the shaft outside the tissue. Local anesthetic can be infiltrated in the area of the barb. Grasp the probe with a clamp and firmly pull traction. A #11 blade can be used to make a small incision in the overlying skin to facilitate the removal of the barb, if needed. Examine the probe after removal to ensure that it was removed intact. These may need to be stored as evidence for law enforcement as dictated by local policy. Perform tetanus prophylaxis and general wound care as indicated.

Patients who have been TASERed by law enforcement are often intoxicated, combative, and suffer from psychiatric illness. It is important to be sensitive to the socially charged nature of these encounters and their medicolegal ramifications. Use objective language in the history and document the source of all information, as histories provided by law enforcement and patients often conflict.

### KEY POINTS

- Routine work-up or observation in the asymptomatic TASERed patient is not necessary if there are no red flags on the history or physical examination.
- An EKG should be performed in symptomatic patients.
- Evaluate for blunt traumatic injuries related to falls, as patients lose protective reflexes during a shock and contraction forces may cause fractures.
- Evaluate for penetrating injuries from the barbed probes, particularly in vulnerable regions (e.g., head, face, genitals, joints, and hands).



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# MANAGING PENETRATING NECK INJURIES: HARD OR SOFT, SUPERFICIAL OR DEEP?

MELISSA JOSEPH, MD

Penetrating neck injuries can range from minor, superficial wounds to life-threatening injuries fraught with significant morbidity and mortality. Workup is minimal in superficial injuries, but deep wounds require further investigation. An algorithmic approach is important to guide evaluation and determine which patients require imaging and surgical exploration.

The division of the neck is important for identifying which underlying structures are at risk for injury, but the classic neck “zones” no longer serve as a hard guide for initial workup.<sup>1–3</sup> (See [Table 258.1](#).) Moreover, many penetrating injuries cross zones.

**TABLE 258.1 ZONES OF THE NECK AND CONTAINED VITAL STRUCTURES**

| NECK ZONE | ANATOMIC BORDER  | STRUCTURES INVOLVED  |
|-----------|--|--|
| Zone 1    | Superior: Cricoid cartilage<br>Inferior: Clavicles and sternal notch | Common carotid arteries<br>Vertebral and subclavian arteries<br>Subclavian, innominate, and jugular veins<br>Recurrent laryngeal and vagus nerves<br>Trachea<br>Esophagus<br>Thoracic duct |
| Zone 2    | Superior: Angle of mandible<br>Inferior: Cricoid cartilage           | Carotid arteries<br>Jugular and vertebral veins<br>Pharynx and larynx<br>Recurrent laryngeal and vagal nerves<br>Spinal cord<br>Trachea  |
| Zone 3    | Superior: Skull base<br>Inferior: Angle of mandible                  | Carotid and vertebral arteries<br>Jugular veins<br>Spinal cord<br>Cranial nerves IX–XII<br>Sympathetic trunk   |

## DETERMINE IF THE WOUND IS SUPERFICIAL OR DEEP

The initial step in the evaluation of a penetrating neck injury (that is not obviously unstable) is to determine whether the injury violates the platysma muscle of the superficial neck. Wounds that do not violate the platysma are unlikely to cause significant injury and do not require further workup.<sup>2,3</sup> Injuries that violate the platysma require early surgical consultation and further investigation.

## INITIATE IMMEDIATE MANAGEMENT IN THE UNSTABLE PATIENT OR WHEN HARD SIGNS OF INJURY ARE PRESENT

Vigilantly monitor the patient for airway compromise, and emergently intervene with evidence of tracheal injury, expanding hematoma, unstable vital signs, or decline in mental status.<sup>1–3</sup> Patients with hard signs of injury (see [Table 258.2](#)) should be taken for surgical exploration and definitive

management.<sup>1–3</sup> Life-threatening injuries most commonly involve damage to the aerodigestive tract and vascular structures.<sup>4</sup>

**TABLE 258.2 HARD AND SOFT SIGNS OF NECK INJURY**

| <b>HARD SIGNS</b>               | <b>SOFT SIGNS</b>                       |
|---------------------------------|---|
| Airway compromise               | Nonexpanding hematoma                   |
| Massive subcutaneous emphysema  | Mild bleeding                           |
| Air bubbling through wound      | Transient, fluid-responsive hypotension |
| Expanding or pulsatile hematoma | Small hemoptysis/hematemesis            |
| Significant active bleeding     | Voice change                            |
| Shock                           | Chest tube air leak                     |
| Neurologic deficit              | Dyspnea                                 |
| Massive hematemesis             |   |
| Pulse deficit                   |   |
| Bruit/thrill                    |   |

## **OBTAIN A CT ANGIOGRAM IN STABLE PATIENTS WITH NO HARD SIGNS OF VASCULAR, AIRWAY, AERODIGESTIVE, OR NEUROLOGIC COMPROMISE**

CT angiography (CTA) of the neck is the modality of choice for evaluation of injury in a patient with penetrating neck trauma that does not otherwise require emergent operative management.<sup>1–3</sup> Patients with soft signs of neck injury (see [Table 258.2](#)) despite a normal CTA, or those with nondiagnostic CTA should undergo further evaluation. Additional tests include magnetic resonance (MR), duplex ultrasound, esophagography or esophagoscopy, and laryngoscopy depending on suspected injury.<sup>1,2</sup>

## **THE ASYMPTOMATIC PATIENT WITH NORMAL CT ANGIOGRAPHY AND NONCONCERNING TRAJECTORY OF WOUND MAY BE DISCHARGED**

CTA has a high sensitivity for detecting significant injury, with one study reporting sensitivity of 100% and specificity of 93.5%.<sup>5</sup> Discharge may be

considered in patients with negative imaging and no hard or soft signs of injury. Those with a concerning trajectory of penetration should be observed with serial examinations or evaluated with additional imaging.

## PITFALLS IN THE EVALUATION AND MANAGEMENT OF PENETRATING NECK INJURIES

- **Potential Pitfall #1: Be meticulous in your examination to evaluate for violation of the platysma and err on the side of caution when uncertain.** While wounds that do not violate the platysma are unlikely to cause injury, those that do can cause significant morbidity and/or mortality. Certain wounds, including puncture wounds, can be deceptively superficial upon first glance. Maintain a low threshold for imaging when the exam is equivocal.
- **Potential Pitfall #2: Be prepared for a difficult airway.** Distortion of anatomy, secretions, blood, and mechanical obstructions may preclude successful rapid sequence intubation. Intubation with ketamine sedation alone may be considered when there is concern that paralysis may further distort anatomy. Initiate a difficult airway algorithm, and be prepared for intervention with a surgical airway.
- **Potential Pitfall #3: Do not unnecessarily place a cervical collar or institute spinal immobilization on a patient with a penetrating neck injury.** Cervical spine immobilization is not necessary in patients who have a normal neurologic examination and mental status.<sup>2</sup> Cervical collars limit full examination of the wound, and spinal immobilization may inhibit airway protection.

### KEY POINTS

- Carefully assess a penetrating neck injury for platysma involvement.
- Vigilantly monitor for airway compromise, and anticipate a difficult airway.
- Initial evaluation of injury in a stable patient is a CT angiogram; symptomatic patients and those with abnormal imaging require further investigation.
- Do not place a cervical collar on a patient with a normal neurologic exam.

- Patients with hard signs of neck injury should be managed operatively.

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# TO CRACK OR NOT TO CRACK: INDICATIONS FOR AN ED THORACOTOMY

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**SANJAY BHATT, MD, MS, MMM**

Few scenarios are as dramatic, time-sensitive, and potentially lifesaving as those requiring an emergency department (ED) thoracotomy. The decision to perform a thoracotomy must be made quickly and without hesitation in the properly selected patient.

## THE BENEFIT OF A THORACOTOMY

An open chest allows visualization of potentially reversible injuries and direct access to intervene. Hemorrhage control in the cardiopulmonary structures can be achieved, cardiac tamponade can be relieved, aortic cross-clamping diverts blood to the heart and improves coronary perfusion, and direct access to internal cardiac massage and defibrillation is provided.

Survival rates range from 2.5% to 27.5% in limited available epidemiologic data. Survival is more likely when signs of life are present in the ED. Nonetheless, there is controversy regarding ED thoracotomy indications given the high cost, low survival rates, low neurologic recovery rates, and the high risk to health care providers.<sup>1</sup>

Under the presumption that an operating room and on-call surgeon are quickly available, guidelines for performance of an ED thoracotomy are summarized in [Table 259.1](#). Most guidelines recommend thoracotomy after the loss of vital signs, although some advocate the procedure in exsanguinating patients who don't respond to initial volume therapy.

Thoracotomy is rarely successful after blunt trauma—potential indications include exsanguination from cardiac or aortic rupture. Indications in penetrating trauma continue to be controversial but generally center on the time from loss of cardiac activity and signs of life.

**TABLE 259.1 INDICATIONS FOR AN ED THORACOTOMY**

|                    | <b>IN-HOSPITAL<br/>LOSS OF<br/>CARDIAC<br/>ACTIVITY</b> | <b>LOSS OF<br/>CARDIAC<br/>ACTIVITY &lt; _X_<br/>MINUTES PRIOR<br/>TO ARRIVAL</b> | <b>HYPOTENSION<br/>DESPITE<br/>AGGRESSIVE<br/>FLUID<br/>RESUSCITATION</b> | <b>&gt;1,500 mL<br/>BLOOD<br/>OUTPUT FROM<br/>CHEST TUBE</b> |
|--------------------|---|---|---|--|
| Penetrating Trauma | Yes   | 15  | Yes   | Yes  |
| Blunt Trauma       | Yes   | 5–10  | Yes   | Yes  |

## CONTRAINDICATIONS TO AN EMERGENT THORACOTOMY

Contraindications include lack of cardiac activity in penetrating trauma 15 minutes prior to ED arrival, lack of cardiac activity in blunt trauma 10 minutes prior to ED arrival, severe head or multisystem injuries, and hospitals with a lack of resources (staff or equipment) to perform the procedure and intervene operatively upon return of circulation.<sup>2</sup>

Survival outside of the indications detailed in [Table 259.1](#) is poor. Ultimately, the decision to perform an ED thoracotomy is made on a case-by-case basis.<sup>3</sup> If stable cardiac activity is regained after a thoracotomy, the patient should be immediately transferred to the operating room for definitive management. Oftentimes, definitive management requires a multidisciplinary approach including trauma, cardiothoracic and vascular surgeons, as well as highly trained support staff.

## COMPLICATIONS OF AN ED THORACOTOMY

Complications include damage to the phrenic nerve (which runs along the lateral surface of the pericardium), neurologic hypoperfusion resulting in anoxic brain injury, damage to the esophagus, and ischemia of the distal organs secondary to hypoperfusion induced by cross-clamping the aorta.

Normal neurologic outcomes occur in the majority of patients who



survive after an ED thoracotomy.<sup>4</sup> Though the indications are limited, there is potential for survival with an excellent outcome. Patient selection is vitally important to achieve optimal outcomes and best utilize resources.

## KEY POINTS

- An ED thoracotomy allows direct access and visualization to the vital organs of the chest and can be a lifesaving procedure.
- The decision to perform an emergent thoracotomy is made on a case-by-case basis, and both the risks and benefits must be weighed.
- In penetrating trauma, an ED thoracotomy can be considered in patients with loss of cardiac activity in-hospital or <15 minutes prior to ED arrival and SBP < 70 despite aggressive resuscitation.
- In blunt trauma, an ED thoracotomy can be considered in patients with loss of cardiac activity in-hospital or <10 minutes prior to ED arrival, SBP < 70 despite aggressive resuscitation, and return of >1,500 mL of blood from a chest tube.

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## PERFORMING AN ED THORACOTOMY

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**SANJAY BHATT, MD, MS, MMM**

A thoracotomy is part of the resuscitation process and must occur in concert with other interventions. One provider should be designated as the resuscitative captain while another performs the procedure. An open chest allows visualization of potentially reversible injuries and direct access to intervene. Hemorrhage control, relief of cardiac tamponade, aortic cross-clamping, evacuation of air emboli, and open cardiac massage are interventions capable of salvaging the patient in extremis after traumatic injury.

Maintain universal precautions with gown, gloves, face shield, and protective footwear. *Potential pitfall: Be mindful of sharps and broken ribs. There is an increased prevalence of blood-borne pathogens in the trauma population, and provider injury is common.* Liberally pour iodine solution over the entire thorax to sanitize the skin. *Potential pitfall: penetrating objects should be left in place unless they interfere with performing the thoracotomy.*

### HOW TO MAKE A THORACOTOMY INCISION

Begin at the sternal edge, and make a generous left anterolateral incision with a no. 10 blade at the left 4th or 5th intercostal space. This incision should be deep enough to expose the intercostal muscle and extend from the sternum to the left posterior axillary line. Make the incision along the upper rib margin to avoid the neurovascular bundle. In women, retract the breast superiorly and incise beneath the breast tissue. *Potential pitfall: avoid the intercostal neurovascular bundles inferior to the ribs.*

Using Metzenbaum scissors, make a small opening through the intercostal muscles into the pleural space. Cut through the intercostal muscles along the upper rib margin.

## HOW TO PROPERLY EXPOSE THE ORGANS

After entering the chest, a rib spreader (Finochietto) is inserted between the ribs with the arm of the retractor directed toward the left axilla. This permits the extension of the incision across the sternum for additional exposure. If needed, the incision can be extended across the sternum to the right with a Gigli saw or Lebsche knife. This clamshell incision improves access to the anterior mediastinum, aortic arch, and great vessels. *Potential pitfall: Control bleeding from the internal mammary arteries after the sternum is divided to prevent iatrogenic hemorrhage.*

## DAMAGE CONTROL IS THE INITIAL PRIORITY

Immediate management upon entering the chest is to control bleeding with direct pressure or a clamp.

## INDICATIONS AND TECHNIQUE FOR PERICARDIOTOMY

The pericardium is typically opened to deliver the heart from tamponade. If there are other obvious injuries present and no evidence of pericardial tamponade, this step may be skipped, although many authors still advocate the opening of the pericardium as accumulations of blood can be deceptively difficult to see.

To perform a pericardiotomy, grasp the pericardial sac with forceps and make a small incision with a knife or Metzenbaum scissors. Extend the pericardiotomy gently parallel and medial to the phrenic nerve. *Potential pitfall: The left phrenic nerve adheres to the anterolateral surface of the pericardium and can be inadvertently injured during pericardiotomy.* Deliver the heart from the pericardial sac and inspect the heart and great vessels for injury. Control rapid bleeding initially with digital compression; a clamped bladder catheter may also be inserted and the balloon inflated for hemostasis. Repair injuries to the heart using nylon suture or staples.

## INDICATIONS AND TECHNIQUE FOR AORTIC

## CROSS-CLAMPING

Cross-clamping the aorta redistributes available intravascular blood volume to the myocardium and brain resulting in a doubled mean arterial pressure and cardiac output. Additionally, potential blood loss from abdominal/pelvic injuries is minimized. *Potential pitfall: cross-clamping the aorta prior to controlling bronchovenous fistulas and removing residual air may result in devastating air emboli.*

Retract the left lung superiorly and divide the inferior pulmonary ligament. Palpate the orogastric or nasogastric tube to help differentiate esophagus from aorta. The aorta is just anterior to the vertebrae, while the esophagus is found anteromedial to the aorta. Clamp the aorta just above the diaphragm.

## OPEN CARDIAC MASSAGE

Cardiac massage should begin immediately after the aorta is clamped and the heart has filled. Aggressive simultaneous volume resuscitation will help fill the heart.

To perform internal cardiac massage, cup the heart with the wrists at the cardiac apex. Squeeze the pericardium using a rhythmic “clapping” motion. *Potential pitfall: Keep thumbs next to index fingers to avoid inadvertent puncture of the heart.*

## INTERNAL DEFIBRILLATION

If ventricular fibrillation is present (diagnosed by direct visualization), internal defibrillation should be performed. Place one internal defibrillation paddle anteriorly and the other posteriorly on the heart. Apply 10 J of defibrillation, and escalate to 50 J as needed.

If resuscitation is successful, patients should be emergently transferred for operative repair of injuries with a multidisciplinary team (trauma, vascular, and cardiothoracic surgeons).

### KEY POINTS

- Emergent thoracotomy should be performed in trauma when therapeutic maneuvers are indicated to manage correctable causes of shock (decompressing cardiac tamponade, cross-clamping the aorta,

managing exsanguinating cardiac or vascular injuries, and evacuating air embolism).

- Hemostasis is the first priority in a thoracotomy to allow for visualization of injuries.
- Pericardiotomy should be the next priority if there is pericardial tamponade followed by hemostasis of cardiac injury.
- Aortic cross-clamping improves cardiac and cerebral perfusion and minimizes bleeding below the diaphragm.
- Open cardiac massage should be initiated after aortic cross-clamping.

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# **SAVE A LIMB! VASCULAR INJURY IN PENETRATING EXTREMITY TRAUMA**

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**TAYLOR McCORMICK, MD**

Extremity injuries may be initially overlooked during the resuscitation of the multiple trauma patient with significant head, neck, thoracic, and abdominal injuries. However, there is significant potential morbidity and mortality in the limbs. This potential exists in a patient with multiple gunshot wounds but also with a single stab wound. Life- and limb-threatening vascular injuries should be recognized, hemostasis achieved, and subtle arterial injuries (which may result in delayed thromboembolic complications) identified. Recognition and management priorities in penetrating extremity trauma are highlighted below.

## **EXTREMITY VASCULAR INJURIES CAN BE SUBTLE**

Vascular injuries can be occlusive, partially occlusive, or occult and include complete and partial transection, acute or delayed thrombosis, reversible arterial spasm, arteriovenous fistula formation, pseudoaneurysm formation, and intimal flaps with subsequent thrombus formation. The path of injury is typically predictable in stab wounds, and structures at risk can be anticipated depending on weapon trajectory. In contrast, damage from gunshot wounds is less predictable due to high-velocity concussive forces and bony ricochet.

## PRIORITIZE HEMORRHAGE CONTROL

Hemostasis is paramount and is best achieved by direct pressure. Tourniquet application may be used when direct pressure fails or in a resource limited setting. Complication rates are low when tourniquets are applied correctly and provide essential hemostasis in transit to the hospital or while awaiting definitive operative repair.

## HARD SIGNS FOR VASCULAR INJURY REQUIRE SURGICAL EXPLORATION

Surgical exploration in <6 hours after injury maximizes limb salvage in patients with “hard signs” of vascular injury (see [Table 261.1](#)). Over 90% of patients with hard signs will have a significant arterial injury requiring repair. Intraoperative angiography or preoperative computed tomography angiography (CTA) may be reasonable in a stable patient with hard signs for operative planning for complex injuries (e.g., shotgun/multilevel gunshot wounds or mangled limb).

**TABLE 261.1 HARD AND SOFT SIGNS OF VASCULAR INJURY**

### **HARD SIGNS**

- Absent distal pulses
- Active pulsatile hemorrhage
- Large expanding hematoma
- Bruit or thrill

### **SOFT SIGNS**

- Distal nerve deficit
- Diminished pulses
- Nonexpanding hematoma
- History of pulsatile or significant hemorrhage at time of injury

## EVALUATE A PATIENT FOR SOFT SIGNS OF VASCULAR INJURY

Patients with penetrating extremity trauma without hard signs of vascular injury should be examined for “soft signs” (see [Table 261.1](#)), and an ankle-brachial index (ABI), or arterial pressure index (API), should be measured. Approximately 25% of patients with soft signs or abnormal ABI/APIs will have vascular injuries requiring surgical intervention. Patients without hard or soft signs of vascular injury and an ABI/API > 0.9 may be discharged.

## **OBTAIN A CT ANGIOGRAM IN PATIENTS WITH SOFT SIGNS OF VASCULAR INJURY OR ABNORMAL ABI/API**

Patients with soft signs of vascular injury or ABI/APIs < 0.9 require further evaluation with advanced imaging. CTA has become the imaging modality of choice, as sensitivity and specificity are comparable to conventional angiography with less patient risk. CTA does not require an arterial puncture or on-site interventional radiologist, is quick and readily available, uses less contrast, and provides detailed anatomy of adjacent structures. Duplex Doppler is less sensitive but may provide information when CTA is not available or if the patient has a contrast allergy.

## **THERE IS CONTROVERSY REGARDING PROXIMITY WOUNDS**

Both the definition (variably defined as 1 to 5 cm from a major neurovascular bundle) and evaluation of proximity wounds are controversial. Some argue proximity wounds are a soft sign. The best available evidence suggests that in patients with a proximity injury, without hard or soft signs and a normal ABI/API, CT angiography is only indicated if the injury was sustained by a shotgun.

### **KEY POINTS**

- Penetrating extremity trauma has great potential for morbidity and mortality. Examine each patient thoroughly for hard and soft signs of injury and perform an ABI/API.
- Hemorrhage control is a priority in penetrating extremity trauma. Tourniquets are an effective adjunctive therapy when direct pressure is not possible or inadequate.
- CT angiography should be performed for patients with soft signs or abnormal ABI/APIs.
- Hard signs warrant surgical exploration.
- Patients with a normal physical exam and ABI/APIs > 0.9 can be discharged home.



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# JUDICIOUS ABDOMINAL IMAGING IN TRAUMA

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**ERICK A. EITING, MD, MPH, MMM**

The evaluation and management of the trauma patient have evolved with advancement of technologies such as computed tomography (CT) and bedside ultrasound. Negative advanced imaging is reassuring and enables us to discharge patients earlier from the emergency department (ED). Previously, many of these same patients required prolonged observation or were taken to the operating room for exploration.

With CT readily available, it should be used judiciously. There are two important pitfalls to be avoided—first, the potential for overutilization and excessive radiation exposure, and second, the drive to obtain imaging studies in critically ill patients that leads to delays in operative management and definitive care. The indications and risks versus benefits of imaging must be carefully weighed to avoid these two potential pitfalls.

## IMAGING INDICATIONS BASED ON MECHANISM OF INJURY

A patient is at risk for different injury patterns depending on mechanism of injury. The focused assessment with ultrasound for trauma (FAST) exam can quickly provide pertinent information that guides management in both blunt and penetrating trauma. While the FAST exam may only have about 40% sensitivity, it has about 99% specificity with an experienced user, with a positive predictive value of 94% and negative predictive value of 95%.<sup>1</sup>

### ***Blunt Trauma***

The physical exam is relatively reliable in awake, alert patients who have sustained blunt abdominal trauma and who do not have neurologic injury. Indications for imaging include significant abdominal tenderness, evidence of peritonitis, referred pain, a positive FAST examination, gross hematuria, or an abdominal “seat-belt sign.” Tenderness in the lower rib cage can be associated with significant liver, splenic, or renal injuries. Referred pain to the shoulders can also signify diaphragmatic irritation due to viscous injury and accumulation of blood. Serial abdominal exams can help identify patients with continued bleeding or viscous injury causing peritonitis. A “seat-belt sign” is also associated with significant abdominal injuries, including spinal and bowel injuries.

### ***Penetrating Trauma***

The mechanism of injury and stability of the patient dictate indications for imaging in penetrating trauma. Gunshot wounds are created at high velocity and cause significant injury to surrounding tissues. Abdominal CT can be informative but should be reserved for stable patients deemed to have a lower likelihood of life-threatening injury. Most of these patients will require laparotomy, and imaging studies should not delay definitive operative management. Abdominal CT should be performed when there is suspicion of violation of the peritoneum. An abdominal CT will also better characterize injuries in hemodynamically stable patients with a positive FAST.<sup>2</sup>

## **IMAGING IN SPECIAL POPULATIONS AFTER ABDOMINAL TRAUMA**

### ***Pediatrics***

Most pediatric trauma is blunt, and the majority of abdominal injuries sustained are managed nonoperatively. Children are most at risk for the consequences of radiation exposure, and CT must be ordered judiciously. Indications for CT in children are similar to those detailed above. US may be helpful to identify injury although the clinician must be aware of the pitfalls of US in the pediatric population. Specifically, the FAST exam may be negative in children with significant intra-abdominal injury, and normal children without injury may have trace pelvic free fluid.<sup>3</sup> The clinical scenario and abdominal examination should guide the decision for CT rather than relying heavily on US.

## ***Pregnant Women***

The FAST examination remains useful in pregnant women who sustain abdominal injury in trauma. Pelvic ultrasound and fetal monitoring can additionally diagnose traumatic obstetric emergencies, such as abruptio placentae, uterine rupture, or fetal injury. However, pregnancy should not preclude appropriate evaluation with CT for potential life-threatening visceral injuries. Discussion of risks and benefits of imaging with pregnant patients is essential when possible.<sup>4</sup>

## ***Geriatric Patients***

In contrast to children, there should be a lower threshold to image elderly patients. Patients over 65 years of age are two to three times more likely to die from trauma than are their younger counterparts. They may initially look well and so are less likely to be admitted to a monitored setting but are at risk for complications secondary to their pathophysiology and associated comorbidities.<sup>5</sup>

Geriatric patients are more prone to bleeding and vascular injuries. They commonly have atherosclerosis and take antiplatelet or anticoagulation medications. Decreased abdominal muscular tone can make patients less likely to develop peritonitis. Older patients also have less general reserve to compensate for acidosis and other manifestations of traumatic injury. Poor nutrition and diuretic medications often leave them dehydrated before trauma, which increases the challenge of resuscitative efforts.<sup>6</sup>

In addition to imaging, careful consideration should be given to extended observation or inpatient admission for the elderly patient after trauma. Additionally, ensuring appropriate social support systems is necessary prior to discharging geriatric patients from the ED.

### **KEY POINTS**

- Patients with a normal mental status, benign abdominal exam, absence of gross hematuria, and without a seat belt sign do not require an abdominal CT after blunt abdominal trauma unless comorbidities are a concern.
- Avoid abdominal CT in patients who are unstable and have clear indications for operative management.

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# SEVERE TRAUMATIC BRAIN INJURY: AVOID MAKING IT WORSE!

ASHKON ANSARI, MD

There is no injury as fatal or as likely to cause long-term neurologic sequelae in survivors as traumatic brain injury (TBI). Not only is TBI the leading cause of death in children and young adults but those that survive often have significant neurocognitive impairment and disability. Although prevention of the *primary injury* is clearly the most effective lifesaving measure, morbidity and mortality can be decreased by vigilantly preventing *secondary injury*.

## CLASSIFYING AND DEFINING INJURY

TBI represents a spectrum of disease ranging from mild concussion to brain death. The Glasgow Coma Scale (GCS) is the most commonly used index of severity score to classify TBI and predict outcomes (see [Table 263.1](#)). The utility of this system is limited in intoxicated patients.

**TABLE 263.1 INJURY SEVERITY SCORE USING GLASGOW COMA SCALE**

| INJURY SEVERITY | GCS   |
|-----------------|-------|
| Mild            | 13–15 |
| Moderate        | 9–12  |
| Severe          | ≤8    |

The initial insult during a traumatic event is the *primary injury*. Mechanisms of injury include blunt force trauma, penetrating injury, blast waves, and rapid acceleration-deceleration. These all lead to direct damage to the brain. Patterns of injury include cerebral contusion, intraparenchymal/intraventricular hemorrhage, extra-axial hematomas (epidural/subdural), traumatic subarachnoid hemorrhage, and diffuse axonal injury.

The acutely traumatized brain is extremely vulnerable to further insult. This is referred to as *secondary injury* and is the focus of the emergency management of TBI. Further injury can be induced by episodes of hypoxia, hypotension, and seizures.

## THE TENETS OF THE EMERGENT MANAGEMENT OF TBI ARE MAINTENANCE OF ADEQUATE OXYGENATION AND CEREBRAL PERFUSION PRESSURE

The greatest detriment to a favorable neurologic outcome is the occurrence of any episode of hypoxia or hypotension—such episodes must be scrupulously avoided.

## KEEP OXYGEN SATURATION > 90% AND VENTILATE TO NORMOCAPNIA (PCO<sub>2</sub> 35 TO 40 MM HG)

In addition to hypoxia, both hypo- and hyperventilation are correlated with worse neurologic outcomes. Endotracheal intubation to protect the airway should be performed early when necessary. This should be performed by the most advanced provider available as even transient hypoxia can further

damage the vulnerable brain. Assessment of the patient's GCS and evaluation of gross neurologic deficits prior to sedation and paralysis is important although intubation should not be delayed in the setting of impending cardiopulmonary arrest.

## **MAINTAIN CPP BY AVOIDING HYPOTENSION AND ENSURING VENOUS DRAINAGE**

Cerebral perfusion pressure (CPP) is equal to mean arterial pressure (MAP) minus ICP. In general, a MAP between 80 and 100 mm Hg will maintain a CPP > 60 mm Hg. Aggressive resuscitation and the identification and treatment of active hemorrhage (including achieving hemostasis from scalp lacerations) is key to maintaining an adequate MAP. Moreover, the single most beneficial technique to reduce ICP (by improving venous drainage) is to elevate the head of the bed to 30 to 45 degrees. Maintain cervical spine precautions as all TBI patients are at risk for concomitant spinal cord injury.

## **GIVE SEIZURE PROPHYLAXIS, AVOID FEVER, AND TREAT COAGULOPATHY**

Seizures can cause acute elevations in ICP and worsen cerebral edema. Although emergent antiepileptic prophylaxis decreases the incidence of early seizures, it does not prevent the later development of epilepsy after TBI. Fever increases ICP, and coagulopathies worsen ongoing bleeding.

## **GIVE OSMOTIC THERAPY WHEN INDICATED**

Osmotic therapy is a temporizing measure to lower ICP. It can forestall brain herniation until more definitive surgical intervention (craniotomy or ventriculostomy) can be performed. Signs of impending herniation include progressive neurologic deterioration and unilateral posturing or mydriasis. Measures to reduce ICP should be initiated as the neurosurgeon prepares for intervention. Mannitol can be given as an intravenous bolus (0.5 to 1 g/kg) but should be used with caution in the polytrauma patient as it may cause hypotension with a resultant decrease in CPP. Hypertonic saline (3% solution or greater) allows for ICP reduction and fluid resuscitation without the risk of compromising CPP. Optic nerve sheath diameter on US has been shown to correlate with ICP and may be an additional tool used to monitor the patient and help guide management.



# ADEQUATELY SEDATE THE PATIENT AND CHOOSE THE AGENT WISELY

Adequate sedation is essential to prevent coughing, agitation, and resultant ICP elevation. Even a brief episode of hypotension can damage a vulnerable brain, so the risks and benefits of drugs used for induction during intubation and for sedation must be carefully weighed. Etomidate is a good choice initially for intubation because of its limited effect on hemodynamic status. Propofol is neuroprotective but may induce unwanted hypotension and should be used in caution in patients at risk for ongoing uncontrolled hemorrhage. Fentanyl decreases ICP but may similarly induce hypotension by attenuating the sympathetic response to trauma. Ketamine is another option; it may improve CPP by increasing MAP and provide some degree of neuroprotection. In patients with structural barriers to normal cerebrospinal flow, however, it may lead to an unacceptable increase in ICP.

## KEY POINTS

- Vigilantly monitor the head-injured patient's respiratory status to avoid hypoxia.
- Aggressively manage concomitant injuries to avoid hypotension.
- Elevate the head of the bed and provide sufficient sedation postintubation to prevent agitation and increased ICP.
- Hypertonic saline is the safest osmotic agent for acutely reducing ICP in patients with polytrauma and impending herniation.

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# FLUID RESUSCITATION IN TRAUMA: FIVE PITFALLS

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ERICK A. EITING, MD, MPH, MMM

Fluid resuscitation in trauma patients is dictated by the patient's clinical status. This begins with the primary survey. Abnormal vital signs; weakened pulses; delayed capillary refill; and cool, clammy extremities all indicate significant blood loss. Tachycardia is often the first manifestation of significant blood loss, followed by a narrow pulse pressure in early shock. The decrease in intravascular volume leads to end organ hypoperfusion, catecholamine release, and increased vascular tone. As blood loss continues, pulse pressure widens, blood pressure drops, respiratory rate may increase, and mental status declines.

Early goals of resuscitation should be focused on identifying and stopping the source of bleeding, maintaining end organ perfusion, treating associated coagulopathies, preventing fluid overload, and facilitating definitive care. Fluid resuscitation should begin with crystalloids (either normal saline or lactated Ringer's). The ideal amount of crystalloid resuscitation is still unknown, and permissive hypotension may be beneficial in trauma. Fluids should be warmed to avoid hypothermia.

## POTENTIAL PITFALL #1: AVOID OVERLY AGGRESSIVE FLUID RESUSCITATION IN TRAUMA

Early, aggressive fluid resuscitation can lead to increased morbidity and mortality. *The focus of resuscitation in major trauma should be on maintaining perfusion and the early initiation of blood transfusion.*<sup>1</sup> Large volumes of crystalloid infusion will cause a dilutional effect on circulating

blood. Patients who require additional volume infusion beyond 1 or 2 L of crystalloid should receive packed red blood cells.

## **POTENTIAL PITFALL #2: DON'T WAIT TOO LONG TO INITIATE A BLOOD TRANSFUSION**

Patients with hemodynamic instability, altered mental status, or significant bleeding should receive blood as soon as possible. Cross-matched blood is preferred to prevent transfusion reactions but is typically not emergently available in the acutely injured patient. Female patients of childbearing age who require emergent blood transfusion should receive O-negative blood, while postmenopausal women and men can receive O-positive blood.<sup>2</sup>

## **POTENTIAL PITFALL #3: FAILURE TO TRANSFUSE MORE THAN JUST PACKED RED BLOOD CELLS**

Utilize a massive transfusion protocol when transfusing >4 to 6 units PRBCs. Ongoing blood loss increases activation and loss of both platelets and coagulation factors. The ideal replacement ratio is as yet unknown, but most massive transfusion protocols have a 1:1:1 ratio of red blood cells:platelets:fresh frozen plasma. The goal is to prevent refractory coagulopathy associated with the depletion of blood products that contribute to clotting.<sup>3</sup> Consider a massive transfusion protocol in patients with (1) penetrating injuries, (2) positive focused abdominal sonography in trauma (FAST) exam, (3) patients who arrive with a systolic blood pressure < 90, and (4) patients with a heart rate > 120.<sup>4</sup>

## **REMEMBER CLOTTING ADJUNCTS AND USE AS INDICATED**

Early tranexamic acid (TXA) improves outcomes if given within 3 hours of injury. Risk increases after the 3-hour window so the time of injury should be known with its use.<sup>5</sup> Aggressively treat coagulopathies, and reverse the effects of anticoagulant medications.

## **POTENTIAL PITFALL #4: RESUSCITATE THE**

## PATIENT BY REPLENISHING FLUID AND BLOOD LOSS RATHER THAN INITIATING VASOPRESSORS

Using vasopressors without adequate fluid resuscitation will only lead to further vasoconstriction and worsening end organ perfusion. Focus on controlling the bleeding, crystalloids, and early transfusion.

## POTENTIAL PITFALL #5: RECOGNIZE AND MANAGE ALTERNATIVE SOURCES OF SHOCK IN AN INJURED PATIENT

Septic or cardiogenic shock may have led to the patient's injury, and neurogenic shock may result after a patient sustained a spinal cord injury. Although hypovolemic shock should always be suspected and treated first, these other causes of shock may play a role in the resuscitation.

### KEY POINTS

- Blood transfusion should be initiated emergently in an ill-appearing or hemodynamically unstable trauma patient.
- A massive transfusion protocol should be initiated in unstable patients and those with significant hemorrhage.

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# **How Do You Fill A Tank With Holes In It? Optimal Vascular Access In Trauma Resuscitation**

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**BENJAMIN D. MUSSER, MD**

Intravenous (IV) access is critical to the management of an injured patient. Access is necessary to infuse fluid, blood products, and medications rapidly enough to compensate for the burden of injury imposed on the patient. Volume resuscitation requires a catheter that is able to infuse large volumes quickly. The ideal catheter for rapid infusion has a wide inner diameter and short length, based on the Hagen-Poiseuille law.

## **How Do I Choose The Correct Intravenous Access In Trauma Resuscitation?**

Several factors determine the optimal IV access in trauma resuscitation. During the initial resuscitation, a large bore IV in each antecubital vein is often sufficient.

## **When Are Peripheral IV Catheters Preferred?**

A 14-or 16-gauge IV placed in a large peripheral vein such as the antecubital vein allows for very rapid fluid administration and is often the optimal IV access in a trauma. Peripheral IV catheters can be placed very rapidly without disrupting the ongoing resuscitation. The largest bore possible should be used, as small increases in the lumen size can drastically increase the flow through the catheter. The main limitation of peripheral access is the restricted ability to administer vasoactive substances and multiple medications simultaneously.

## **WHEN IS A CENTRAL LINE INDICATED?**

Central access is most beneficial when rapid fluid or blood product infusion is necessary, multiple medications are given, and vasoactive agents are needed.

A central venous sheath is the best option in an unstable patient requiring blood products. A large-bore introducer sheath with one single port of 7.0 to 8.5 French allows the extremely rapid infusion of blood products into a patient requiring significant resuscitation. Despite their longer length, when used with pressure bags, large-bore introducer sheaths can reach infusion rates of >800 mL/min. In contrast, large-bore peripheral IV access catheters (14 or 16 gauge) have flow rates that are more comparable to smaller (4 or 5 French) introducers.

Triple-lumen catheters are best when a patient requires administration of multiple medications simultaneously. Multiple lumens can infuse medications and blood products simultaneously.

## **LIMITATIONS OF CENTRAL ACCESS**

Central lines sacrifice the ease and speed of insertion that peripheral IVs command, and they must usually be placed in a sterile manner by an experienced clinician. The triple lumen catheter's increased length results in a decreased flow rate per lumen compared to peripheral access. Additionally, placement of high lines may interfere with resuscitative efforts, especially cardiopulmonary resuscitation (CPR).

## **WHEN SHOULD AN INTRAOSSEOUS (IO) LINE BE USED?**

If peripheral IV access is unsuccessful, intraosseous (IO) lines provide rapid access. They can be placed in under one minute by experienced clinicians.

Infusion in an IO line is much slower with gravity than with peripheral IV access, so a pressure bag or pump must be used in a critical patient. With rapid infusion through an IO line, care must be taken that mechanical complications/extravasation does not occur. Lastly, infusion into bone marrow is painful—so use 2% sterile cardiac lidocaine (0.5 mg/kg) in an awake patient.

## WHERE IS THE IDEAL LOCATION OF ACCESS?

Both the size and location of IV access are important in trauma resuscitation. Despite their pitfalls, high lines allow more rapid distribution to the central circulation. Vessels may be potentially injured in a trauma victim. Vascular access at the site of traumatic injury may compromise the delivery of medication, fluid, or blood products and should be avoided. Access should also be avoided in obviously injured extremities if possible. Femoral central access should also be avoided in significant abdominal and pelvic trauma with potential large vessel damage.

### KEY POINTS

- The optimal IV access in the severely injured trauma patient allows rapid infusion of large volumes.
- A 14- or 16-gauge IV in a large peripheral vein is often the optimal IV access.
- Central venous access allows for rapid resuscitation and administration of multiple medications simultaneously.
- The intraosseous route is a viable alternative in a trauma patient when peripheral access is unsuccessful.
- The site of traumatic injury should be avoided if possible.

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## DON'T BE AFRAID TO PLACE A CHEST TUBE

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**DHARA P. AMIN, MD**

Trauma is the leading cause of death in young individuals. Pneumothoraces occur as a result of rupture of the visceral or mediastinal pleura, allowing air to enter the pleural space. Traumatic pneumothoraces can be caused by penetrating or blunt trauma, be open or closed, and be either simple or tension. In thoracic trauma, the most common intervention is the drainage of air or fluids from the pleural space by means of a chest tube. The thoracostomy procedure can be done at bedside or in an operating room and is the definitive treatment in the majority of cases. In the emergency department (ED), it is performed in life-threatening emergencies.

Chest radiography remains the standard diagnostic test for evaluation of thoracic trauma in the ED. In the upright patient, the classical picture of a fluid level with a meniscus can be seen in the setting of hemothorax. It may take as much as 400 to 500 mL of blood to obliterate the costophrenic angle on a chest radiograph. In the supine position, no fluid level is visible as the blood lies posteriorly along the posterior chest, though, depending on the size of the hemothorax, the chest radiograph may show a diffuse opacification of the hemithorax, through which lung markings can be seen.

The use of point-of-care ultrasound in trauma to the thorax has gradually expanded over recent years. The ability of ultrasound to detect pneumothoraces at the bedside is very valuable indeed, especially when one considers that more than half of all pneumothoraces are missed on a supine chest radiograph. A lung ultrasound is more sensitive than chest radiography for the detection of pneumothorax and can be performed rapidly at the bedside, preventing any delay in diagnosis. Findings consistent with a normal lung-pleural interface are absent and the absence of lung sliding is 95%

sensitive for a pneumothorax. Computed tomography (CT) has the highest sensitivity in detecting pneumothorax or hemothorax, but it is more time consuming and requires a stable patient.

The indications for chest tube placement in trauma can be divided into absolute and relative. The absolute indications are a pneumothorax, hemothorax, or traumatic arrest. The relative indications include positive pressure ventilation with rib fractures and profound hypoxia or hypotension with penetrating chest injury. Patients in traumatic arrest with no cardiac output should have immediate decompression of both sides of the chest to exclude tension pneumothorax. Similarly, patients in shock or profoundly hypoxic with unilateral chest signs or evidence of penetrating trauma to a hemithorax should have a chest drain placed emergently. A chest tube should also be considered for patients about to undergo air transport and who are at risk for pneumothorax.

In the injured patient, a chest tube may be lifesaving, facilitating evacuation (and monitoring) of hemothorax and preventing the development of tension pneumothorax. Chest drainage may also promote lung reexpansion, tamponades low-pressure pulmonary bleeding, and improves respiratory function. Procedural sedation and analgesia in the conscious patient is a critical element of the procedure, with a variety of excellent choices for intravenous (IV) titration available. There is no absolute contraindication to the placement of a chest tube in trauma patients.

Chest tubes placed in emergent settings with an unstable or altered patient are performed without consent as a lifesaving procedure. In all other scenarios, consent should be obtained from the patient and potential risks need to be explained. Minor complications of thoracostomy tube placement such as unresolved/reaccumulation of pneumothorax or misplacement of the tube are common. Other complications include local bleeding or iatrogenic hemothorax by injuring the lung or intercostal artery during placement. There can be injury to the liver or spleen leading to hemoperitoneum if the tube is placed too far inferiorly in the thorax or directed inferiorly after placement, which may necessitate subsequent emergent laparotomy. Infection such as an empyema can develop from the chest tube introducing bacteria into the pleural space if not done in a sterile fashion. Lastly, reexpansion pulmonary edema is a rare and potentially fatal complication that can occur after treatment of pneumothorax or a pleural effusion.

The indications for placing a chest tube are as important as the complications. Explaining to patients both the benefits and the risks is a crucial part of the procedure. Using proper technique for placement and doing it in a sterile fashion when possible can prevent many of the common

complications.

## KEY POINTS

- Chest radiography can miss up to half of all hemo-and pneumothoraces. Ultrasound is more sensitive and ideal in the patient too unstable for CT.
- Chest tubes are indicated in the setting of traumatic hemo-and pneumothoraces, as well as traumatic arrest.
- Placement of chest thoracostomy should be considered in hypoxic or hypotensive patients with chest trauma, in intubated patients with rib fractures, and in patients being transported via air at risk for pneumothorax.

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# MASSIVE TRANSFUSION IN TRAUMA: A CHANGING LANDSCAPE

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**TARLAN HEDAYATI, MD**

The goals in the management of a patient with traumatic hemorrhage are to maintain volume, restore oxygen delivery to vital organs, and prevent additional blood loss via hemorrhage control and correction of coagulopathy. In some cases, the presence of massive hemorrhage is obvious, for example, a hypotensive patient with a brisk external bleeding source. In many cases, however, it is very difficult to predict the onset of hemorrhagic shock in the patient who is currently stable or quasi-stable. There is concern for both underresuscitation (e.g., waiting too long to give blood) and overly aggressive resuscitation (e.g., inappropriate administration of blood products).

Prehospital and emergency department (ED) resuscitation of the bleeding trauma patient traditionally began with infusion of crystalloids, such as normal saline or lactated Ringer's. However, while crystalloid infusion may improve volume status, it also contributes to hemodilution, acidosis, and coagulopathy—all of which may contribute to increased morbidity and mortality in these already critically ill, hemorrhaging patients. More recently, there has been a shift in resuscitation guidelines, with most now advocating the avoidance of large fluid administration in favor of earlier blood product transfusion instead. Ultimately, crystalloid cannot replace the oxygen-carrying abilities of blood.

A massive transfusion (MT) has historically been defined as administration of 10 units or more of red blood cells in a 24-hour period,

transfusion of more than 4 units within 1 hour, or the replacement of more than 50% of a patient's blood volume in a 3-hour period. Massive transfusion protocols (MTP) are institutional algorithms that are developed to coordinate resources such as the blood bank, laboratory, nursing, and pharmacy in the care of the hemorrhaging patient.

Which trauma patients will require MT prior to signs of clinical instability? Studies have demonstrated that "clinical gestalt" is unreliable and a poor predictor of need for transfusion. There are various scoring systems available to help predict which trauma patients will require transfusions, including the assessment of blood consumption (ABC) score, the trauma associated severe hemorrhage (TASH) score, and the traumatic bleeding severity score (TBSS), among others. The simplest, the ABC scoring system, which has been validated prospectively, assigns one point for each of the following: heart rate > 120, systolic blood pressure < 90, a penetrating mechanism, and a positive focused assessment with sonography for trauma (FAST) examination. A score > 2 indicates that the patient will likely need MT. A negative predictive value of <5% means the ABC score identifies 95% of patients requiring a MT. However, a positive predictive value of 50% to 55% means that it also seriously overestimates the need for a MT. Most clinicians will use some combination of the ABC score, persistent clinical instability, active bleeding (clinically or on imaging studies), or multiple transfusions in the ED to activate a MTP.

Transfusion in trauma patients has not been informed by high-quality evidence and research until recently. US military experience led to the proposal of using a 1:1:1 ratio of plasma, platelets, and packed red blood cells in trauma patients requiring MT. The Pragmatic Randomized Optimal Platelet and Plasma Ratios (PROPPR) study, published in 2015, sought to elucidate the ideal ratio of blood product transfusion. The primary outcomes were 24-hour and 30-day mortality. Additionally, the study looked at other prespecified criteria such as time to hemostasis, blood product volumes transfused, and complications. The investigators of this randomized, multicenter trial, found that patients who received a 1:1:1 ratio of these blood products, as compared to 1:1:2 ratio, had no significant difference in mortality, but did have lower rates of exsanguination and higher rates of hemostasis. There was no difference in complication rates between the two groups, but more plasma and platelets were used in the 1:1:1 group. There was no difference in PRBC use between the groups. This has led to a recommendation of using a 1:1:1 ratio of blood products in MTP. Besides the usual risk of transfusion reactions and transmission of disease in patients receiving transfusions, patients undergoing MT are at risk for volume overload, hypocalcemia (due to citrate), hyperkalemia, acidosis, and

hypothermia.

In addition to blood products, MTP may include pharmacologic agents to assist in the control of bleeding. Tranexamic acid (TXA) is a common agent available for use in the ED to help stanch the flow in the hemorrhaging patient. It is an antifibrinolytic agent that inhibits the activation of plasminogen to plasmin. Plasmin, in turn, is responsible for the breakdown of fibrin. The role of TXA in the hemorrhaging trauma patient was investigated in the Clinical Randomisation of an Antifibrinolytic in Significant Haemorrhage (CRASH-2) study, an international, randomized, controlled trial of over 20,000 trauma patients with or at risk for significant bleeding. The primary outcome was death within 4 weeks of injury. TXA, given intravenously as a bolus of 1 g over 10 minutes followed by 1 g over 8 hours, significantly reduced all cause mortality and death due to bleeding as compared to placebo, without any increase in vasoocclusive events or deaths. To exert its positive effects, TXA should be given within the first 3 hours after trauma.

## KEY POINTS

- Excessive crystalloid administration should be avoided in the hemorrhaging trauma patient.
- Predicting who needs MT remains difficult, but evidence supports a blood product transfusion ratio in a MTP is 1:1:1, plasma, platelets, and packed red blood.
- TXA should be considered as an adjunct to transfusion in the hemorrhaging patient.

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## REVERSAL OF WARFARIN IN TRAUMA

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Warfarin sodium (Coumadin®) is the most common oral anticoagulant in the United States. The aim of warfarin therapy is to decrease morbidity and mortality by reducing thrombus burden and limiting pathologic sequelae of excess thrombus formation.

Warfarin works by inhibiting vitamin K epoxide reductase, preventing the liver's inherent ability to recycle existing vitamin K. This reduces the production of coagulation factors II, VII, IX, and X (the vitamin K–dependent factors), ultimately reducing fibrinogen conversion to fibrin. The degree of anticoagulation is measured by the international normalized ratio (INR). Normal values range from 0.8 to 1.2, while those taking warfarin have goal INRs ranging from 2.0 to 3.5 depending on their indication for anticoagulation. INRs above 4.0 are considered supratherapeutic; such patients are at significantly increased risk for bleeding, both spontaneously and related to trauma.

In trauma care, where hemorrhage control remains a cornerstone of therapy, patients anticoagulated with warfarin further complicate hemostasis and pose additional challenges in treatment and disposition. Although several options exist for the reversal of warfarin anticoagulation, none is without their own potential drawbacks.

Vitamin K (phytonadione) overcomes warfarin by providing increased substrate for the formation of vitamin K–dependent coagulation factors. While available in oral (PO), subcutaneous (SQ), or intravenous (IV) formulations, only IV administration is recommended for serious bleeding. Oral administration is acceptable for patients with a supratherapeutic INR in

minor trauma without evidence of bleeding. Subcutaneous administration is not recommended due to the unpredictable rate of absorption. While the 2-hour time of onset limits the rapid benefit of anticoagulation reversal, vitamin K is still recommended to prevent rebound bleeding later on. Rapid administration of IV vitamin K is associated with an anaphylactoid reaction; it is therefore recommended that the IV formulation be dispensed as a “slow IV push” at a rate not faster than 1 mg/min.

Administration of fresh frozen plasma (FFP) replenishes low levels of vitamin K–dependent coagulation factors and allows for increased activation of the coagulation cascade. As a blood product, FFP necessitates blood type matching to avoid serious transfusion reactions and patient consent, when possible. Prior to administration, FFP must be thawed 30 to 45 minutes before it is ready for use. Both these factors limit the immediate availability of FFP. Some blood banks circumvent the thawing time by storing prethawed FFP, but this comes at a cost of decreased shelf life.

The appropriate dose of FFP for warfarin reversal is 10 to 15 mL/kg. This can be a significant fluid bolus to the anticoagulated patient, who is typically elderly and more sensitive to volume overload. Despite these limitations, FFP remains the most commonly used replacement of coagulation factors, though newer therapies are emerging in the market as alternatives.

Prothrombin complex concentrate (PCC) is the pooled plasma product of vitamin K–dependent coagulation factors II, VII, IX, and X. Available in 3-factor PCC (lower levels of factor VII) and newer 4-factor PCC (higher levels of factor VII) formulations, PCC is immediately available for administration, shelf stable and low in volume. Multiple studies demonstrate both an increased rate of INR normalization and decreased time to normalization when compared to FFP. While PCC is more effective at correcting INR, it may come at the cost of increased thromboembolic events. Early studies with 3-factor PCC found increased incidents of deep vein thrombosis and myocardial infarction compared to plasma, but more recent studies with 4-factor PCC show no difference in thromboembolic events.

Recombinant factor VIIa (rFVIIa) is a synthetic blood product, initially developed for the treatment of hemophilia but now available for off-label use in warfarin reversal. Despite early studies that showed promise, no studies to date have shown that rFVIIa improves outcomes compared to standard therapy. It does, however, result in an increased risk of thromboembolic events and is currently very expensive.

Minor head trauma in the anticoagulated patient deserves special mention. While no seminal research exists demonstrating the absolute need

to obtain a head computed tomography (CT) for all of these patients, most clinicians are very liberal with imaging. While a positive CT (e.g., acute bleeding) necessitates full INR reversal, negative imaging can provide a disposition dilemma. Several studies have highlighted a risk for delayed bleeding. Delayed bleeding occurs in a reported 0.5% to 6.0% of cases, although it remains unclear what percentage of these are clinically significant. Most experts recommend reversal of any *supratherapeutic* INR, a period of observation and consideration for repeat imaging. More specific recommendations than these are highly variable, and management should take into consideration both the severity of the trauma and the condition of the patient.

## KEY POINTS

- Traditional reversal of warfarin therapy in the setting of trauma includes transfusion of fresh frozen plasma (10 to 15 mL/kg) and IV vitamin K administration.
- PCC may be more effective than FFP at reversing warfarin, but it is more expensive and may be associated with more thromboembolic events.
- Delayed bleeding does occur in anticoagulated patients in the setting of head trauma. Observation and good aftercare instructions are essential to avoid missing these cases.

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# REVERSAL OF NOVEL ANTICOAGULANTS AND ANTIPLATELET AGENTS

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In recent years, novel anticoagulant (NOAC) and antiplatelet medications have been approved and are being prescribed with increased frequency. With increased usage, these agents create new challenges for the emergency management of bleeding patients. Knowledge of drug names, their mechanism of action, and, most importantly, management of complications and possible reversal methods is crucial for those in the emergency department (ED).

The numerous anticoagulants and antiplatelet agents are classified based on their mechanism of action. Argatroban, bivalirudin, and lepirudin are all intravenous direct thrombin inhibitors (DTIs) and have short half-lives that range from 25 to 80 minutes. These medications are infrequently initiated in the ED, and, therefore, complications in the ED are rare. Emergent management of hemorrhagic complications is primarily supportive and begins with interruption of treatment, transfusion of packed red blood cells as needed, and transfusion of fresh frozen plasma (FFP). No specific reversal agent is available for these drugs. Dabigatran is an oral DTI that is rapidly but poorly absorbed by the GI tract. If the patient requires reversal within 2 to 3 hours of ingestion, activated charcoal should be given as the drug is lipophilic and charcoal should prevent absorption. Hemodialysis is effective at removing ~60% of the drug. Some animal and human studies have shown that the effects of dabigatran can be reversed with activated prothrombin complex concentrate (aPCC), though this would currently be an off-label use of these products. aPCC contains the vitamin K-dependent factors II, VII,

IX, and X in partially activated form. Idarucizumab has been approved by the U.S. Food and Drug Administration (FDA) specifically for the reversal of dabigatran in emergency or life-threatening conditions. However, further studies are needed to determine its duration of reversal and impact on patient outcomes.

Apixaban, rivaroxaban, and fondaparinux are direct factor Xa inhibitors and have no specific reversal agent; management is, again, only supportive. Moreover, hemodialysis is ineffective for these agents due to their high plasma protein binding capacity. Prothrombin complex concentrate (PCC) and aPCCC have been shown to reverse the anticoagulation effects of apixaban and rivaroxaban in some animal and human studies, though this currently represents an off-label use of the product. There is no role for vitamin K in the reversal of any of the NOACs.

Antiplatelet agents available in the United States that irreversibly inhibit platelet function include aspirin, clopidogrel, prasugrel, and ticagrelor. Duration of platelet inhibition is not dependent on the agents' half-life, but may persist for 5 to 7 days. There are no specific reversal agents for antiplatelet agents. Management of antiplatelet agent-associated bleeding events includes discontinuation of the antiplatelet agent. Stopping the drug due to a bleeding event must be weighed against the patient's risk of arterial thrombosis. Platelet infusion may be considered as an additional measure for severe bleeding or prophylaxis before emergency surgery, but again may confer a risk of thrombosis. Another reversal adjunct is desmopressin (DDAVP), though it can also lead to arterial vasospasm.

The use of NOACs and new antiplatelet agents for the treatment of atrial fibrillation, acute deep venous thrombosis, coronary artery disease, and stroke has increased despite the lack of known antidotes to most of these new medications. The advantages over traditional warfarin therapy include rapid onset, fewer drug and food interactions, no lab monitoring, and near-equivalent rates in prevention of stroke and thromboembolism. Bleeding rates are similar, although arguably much more dangerous given the limited experience we have treating bleeding complications.

No matter the agent, the approach to bleeding in the anticoagulated patient involves going back to the basics. Discontinuation of the agent, compression at the site of bleeding, and fluid replacement/transfusion of blood are all simple tactics that work. Hemodialysis works only for dabigatran, but there may be a role for aPCCC and PCC for the other agents. Fortunately, help is on the way. There are specific reversal antidotes currently being studied that may revolutionize the management of bleeding complications in patients on NOACs and antiplatelet agents.

## KEY POINTS

- Initial management of life-threatening bleeding in patients on NOACs and antiplatelet agents includes cessation of the medication, compression at the bleeding site if possible, packed red blood cell transfusion to correct current or potential anemia, and appropriate plasma and platelet transfusion.
- Idarucizumab is FDA approved for the reversal of dabigatran in emergency or life-threatening conditions. None of the other drugs have specific reversal agents.
- There may be a role for aPCC and PCC in the management of bleeding patients on direct factor Xa inhibitors (apixaban, rivaroxaban, and fondaparinux).

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## WHEN TO SUSPECT CERVICAL VASCULAR INJURY

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The patient that sustains blunt trauma is at risk for cervical vascular injuries, specifically the carotid, subclavian, and vertebral arteries, as well as the internal and external jugular veins. Up to one-third of patients with blunt cervical vascular injuries have multivessel injuries involving more than one carotid or vertebral artery.

Blunt carotid injury has a low incidence (0.24% to 0.33%) among trauma patients but a high morbidity (up to 58%) and mortality (up to 33%), with mortality most commonly due to stroke. The carotid is most commonly injured via hyperextension or stretching of the vessel, direct blow, or laceration from bony fragments. Vertebral artery dissection can occur with even “minor” trauma such as with chiropractic manipulation or yoga. They are frequently associated with cervical spine fractures. With blunt cervical injury, there is not typically frank vascular disruption and exsanguination; instead, dissection, thrombosis, embolization, or pseudoaneurysm formation is more typical. The classic clinical scenario associated with cervical vascular injury is that of a trauma patient with hemispheric neurologic defects or decreased level of consciousness in the face of an initially negative noncontrast computed tomography (CT) of the head. However, signs and symptoms of a cervical vascular injury may not be apparent acutely; 17% to 35% of patients with traumatic carotid dissection do not have any symptoms for 24 hours. This latent phase delays diagnosis and treatment, thereby increasing morbidity and mortality.

The Modified Denver Screening Criteria are a collective set of signs and



symptoms developed to assist the emergency physician to screen for a cervical vascular injury in the setting of blunt trauma. These signs include (1) arterial hemorrhage, (2) carotid bruit, (3) expanding cervical hematoma, (4) focal neurologic deficit, (5) neurologic findings that are not explained by neurologic imaging, and (6) ischemic stroke on secondary head CT. Additionally, the following injuries are also highly associated with cervical vascular injuries: cervical spine fracture, Le Fort II or III fracture, basilar skull fracture involving the carotid canal, near-hanging with anoxic brain injury, and diffuse axonal injury with Glasgow Coma Score (GCS) < 6. A patient with a high force injury and any of the aforementioned signs or injuries should undergo diagnostic imaging to rule out vascular injury.

The traditional reference standard for imaging of the cervical vasculature was digital subtraction angiography (DSA), with a sensitivity and specificity approaching 100%. However, DSA is costly, invasive, not readily available due to personnel and equipment requirements, and associated with complications including dissection, thrombosis, embolization, vasospasm, and renal failure. DSA also has a stroke complication rate of 0.1% to 1%. CT angiography (CTA) has largely replaced DSA due to its availability, speed, cost-effectiveness, and ability to evaluate for other cervical structures and injuries. Sensitivities of 97% to 100% and specificities of 94% to 100% for vascular injury have been reported. Disadvantages to CTA include streak artifacts from metallic foreign bodies such as dental fillings or previous cervical spine surgeries, difficulty visualizing vessels running through bone, radiation, and contrast toxicity. Magnetic resonance angiography (MRA) is another option but, like DSA, has limited availability, may not be an option in the acute trauma setting due to its remote locale, and is contraindicated in patients with many types of metallic foreign bodies. Moreover, due to the long image acquisition times, it is not a practical option in a multisystem trauma patient who requires rapid imaging of the chest, abdomen, and pelvis. Furthermore, hematoma on MR in its early stages may appear isodense to surrounding structures and make it difficult to identify acute dissection. Sensitivities of MRA range between 50% and 100% and specificities between 29% and 100%. At this point, most guidelines recommend the use of CTA for screening purposes and reserve DSA for the cases where CTA findings are equivocal.

## KEY POINTS

- Patients with blunt cervical vascular injury may not present initially with neurologic or vascular signs and symptoms.

- Both major mechanisms (cervical fractures) and seemingly minor ones (chiropractic manipulation) may cause cervical vascular injury.
- Signs, such as hematoma or bruit over the carotid area, high-risk mechanisms (e.g., near hanging), or neurologic findings (stroke) should all prompt imaging of the cervical vessels.
- CTA is the diagnostic screening modality of choice. DSA should be done only if CTA is equivocal.

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## ALTERNATIVES TO PACKED RED BLOOD CELLS: THE LATEST

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Current practice in the United States calls for replacement of lost blood with banked allogeneic blood products and/or fluids in the setting of hemorrhage. The quest for a successful blood substitute has been ongoing for nearly a century. The prospects of eliminating blood shortages, transmission of transfusion-related diseases, and antigenic transfusion reactions have fueled research and attempts to develop a suitable substitute. Unfortunately, there are currently no oxygen-carrying blood substitutes approved by the Food and Drug Administration (FDA).

In the emergency department (ED), lost blood volume is typically initially replaced with crystalloids, such as normal saline or lactated Ringer's. However, while fluids may replace volume and improve blood pressure, they are an inadequate substitute for blood; they cannot replace hemoglobin's oxygen-carrying capacity. Multiple studies have demonstrated that trauma patients who receive aggressive fluid volumes tend to have worse outcomes than do those who receive less fluid or receive blood products earlier. Aggressive crystalloid administration leads to hemodilution, volume overload, acidosis, coagulopathy, and hypothermia.

Hypertonic saline can be used in the setting of traumatic brain injury or elevated intracranial pressure. Other options for volume replacement include nonprotein colloids, such as hydroxyethyl starch (HES), albumin, dextrans, and gelatins. HES, a synthetic colloid, has not been shown to be superior to crystalloid and is associated with worsening renal function and acute kidney injury, thought to be due to increased glomerular filtration of the hyperoncotic colloids. Similar rises in creatinine are seen in patients receiving dextrans. Gelatins can cause anaphylaxis, hyponatremia, and

hypotension secondary to release of bradykinins. Albumin, harvested from human plasma, is a protein colloid, which again has not been shown to be clinically superior to crystalloid as a volume expander, has been associated with increased mortality in some studies, is expensive, and has limited availability. Albumin has been associated with hypotension if infused rapidly, acute congestive heart failure in those patients at risk for it, and the potential transmission of prions. It should be considered a “third choice” after crystalloids and nonprotein colloids have been used at maximum dosages.

Autologous blood transfusion, or the collection and reinfusion of a patient’s own blood, is a very feasible alternative to transfusion of donor blood. This is particularly useful prior to a surgical procedure if the potential need for blood is identified ahead of time. Packed red cells have a storage life of about 35 days, allowing for multiple units of blood to be collected and stored prior to a procedure. In the ED, autologous blood transfusion is typically done on patients with hemothorax requiring tube thoracostomy, using a collection device specifically for autologous transfusion. Advantages of autologous transfusion over donor PRBC are multiple: the blood is immediately available; carries no risk of transmissible disease or incompatibility reaction; is already warm; has better oxygen-carrying capacity; does not produce electrolyte abnormalities such as hypocalcemia, hyperkalemia, or acidosis; contains platelets and clotting factors; and is acceptable to patients with religious or other personal objection to allogeneic blood transfusion. Risks of autologous transfusion include infection secondary to contamination, particularly in patients with penetrating thoracic injuries and air embolus.

Lastly, in chronic conditions such as anemia due to chronic kidney disease, chemotherapy, and antiretroviral agents, the use of growth factors such as epoetin alpha can be used to increase hemoglobin. It takes several weeks to see a hemoglobin rise from epoetin use, and it is, therefore, not an appropriate alternative to blood transfusion in patients with symptomatic anemia or acute hemorrhage.

## KEY POINTS

- There are currently no commercially available and approved oxygen-carrying blood substitutes.
- When possible, autologous blood transfusion and cell salvage should be used to replace lost red blood cells.

- Initial volume expansion can be achieved with crystalloids, but crystalloids do not replace the oxygen-carrying capacity of blood and are associated with hemodilution, volume overload, coagulation derangement, and acidosis.

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## **TOUGH BREAK: ASSESSING AND TREATING RIB FRACTURES**

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**MICHAEL GOTTLIEB, MD, RDMS**

Rib fractures may be associated with direct trauma, as well as more benign processes (e.g., excessive coughing). Rib fractures should be suspected in any patient with localized pain, tenderness, or crepitus on palpation of one or more ribs. Conventional radiography has been demonstrated to miss over 50% of rib fractures. Despite that, isolated rib fractures are of minimal clinical significance as long as the following aspects are addressed.

The most important consequence of rib fractures is the potential for underlying injury. One must be careful to assess for associated pulmonary contusions, pneumothoraces, hemothoraces, and intra-abdominal injuries. Most of these can be readily identified on the initial chest radiograph, but in certain cases, chest computed tomography (CT) or ultrasound (US) may be necessary to supplement the initial imaging. Isolated rib radiographs are of minimal utility. Rib fractures can also be very painful, which may interfere with adequate ventilation due to splinting, resulting in atelectasis and pneumonia.

Immobilizing the chest wall with tape or binders is no longer recommended as it has been demonstrated to promote hypoventilation. Pain due to rib fractures can be difficult to manage. Typically, a combination of nonsteroidal anti-inflammatory and opioid medications will be necessary along with incentive spirometry to prevent atelectasis. Lidocaine patches placed directly over the rib fracture are also a useful analgesic adjunct. Care should be taken not to place the patch directly over broken skin. For patients requiring more significant analgesic doses or for whom these medications are contraindicated, an intercostal nerve block or epidural catheter may be preferable.

One exception to the above recommendations is the presence of flail chest. Flail chest is defined as the presence of two or more rib fractures in three or more adjacent ribs and may be identified by the paradoxical inward movement of the involved portion of the chest during inspiration. These patients should be closely monitored with a low threshold for intubation and mechanical ventilation in the presence of respiratory difficulty.

Most rib fractures heal uneventfully within 3 to 6 weeks. Patients with multiple fractured ribs, with preexisting pulmonary disease, or of advanced age should be considered for admission for 24 to 48 hours.

## KEY POINTS

- Be sure to assess for underlying complications of rib fractures, such as pulmonary contusions, pneumothoraces, hemothoraces, and intra-abdominal injuries.
- Ensure adequate pain control.
- Encourage incentive spirometry.
- Consider admission for the elderly, patients with preexisting pulmonary disease, and those with multiple rib fractures.

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## NOT SO FAST: PEARLS AND PITFALLS WITH THE FAST EXAM

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**MICHAEL GOTTLIEB, MD, RDMS**

Focused assessment with sonography in trauma (FAST) is one of most commonly performed point-of-care sonographic procedures. Initially described in the early 1990s, there has been tremendous research and training on the FAST examination, and it is now considered a core sonographic examination in emergency medicine. The typical FAST examination includes a right upper quadrant (RUQ) abdominal view, left upper quadrant (LUQ) abdominal view, suprapubic view, and subxiphoid or parasternal cardiac view. There are a number of pearls and potential pitfalls with this procedure.

The most common initial FAST view is the RUQ view assessing for fluid in Morison's pouch (hepatorenal recess). It is important to thoroughly fan the probe all of the way in each direction, as small slivers of intraperitoneal fluid can easily be missed with incomplete visualization. This can be difficult in patients with small rib spaces and may require moving the probe up or down one rib space to allow adequate visualization. If fluid is identified in the RUQ, it is important to differentiate true intraperitoneal fluid from false positives, such as perinephric fluid, bowel, or renal cysts. These can best be differentiated by scanning all of the way through any suspected fluid when detected. Bowel will demonstrate peristalsis if the probe is left in place for 3 to 5 seconds. Perinephric fluid and renal cysts can be differentiated by identifying Gerota fascia, a highly echogenic line surrounding the kidney. Intraperitoneal fluid will be located between this line and the liver, while perinephric fluid and renal cysts will be located between this line and the kidney.

The LUQ view may be more difficult to perform as the provider is often required to place the probe more posterior and superior, which can result in



narrower rib spaces. One common pitfall with this view is the failure to visualize the splenophrenic (infradiaphragmatic) space. In the RUQ, the liver abuts the diaphragm, so it is uncommon for fluid to accumulate above the liver. However, in the LUQ, there is a large potential space between the spleen and diaphragm, and, in the supine patient, this is a common place for free fluid to collect.

The suprapubic view is much more accessible than the prior two views but may be challenging in patients without a full bladder due to loss of the acoustic window. Consider instilling sterile normal saline into the bladder if a catheter is already in place to recreate the acoustic window. It is also important to note that women of childbearing age may have a small amount of physiologic fluid present. Preexisting ascites or iatrogenic fluid (e.g., in patients receiving peritoneal dialysis) may also result in false positives.

Cardiac visualization, assessing for pericardial effusion or tamponade, may be obtained using either a subxiphoid or parasternal long approach. The subxiphoid view is preferred in thinner patients or those with COPD, as the thin rib spaces and hyperaerated lungs may significantly interfere with obtaining good parasternal views. In the obese patient or those with abdominal pain, the subxiphoid may be difficult to obtain and the parasternal view is preferred. One common pitfall with this exam is mistaking pleural fluid for pericardial fluid on the parasternal long view. These may be differentiated by identifying the descending aorta. Pericardial fluid will be located anterior to the descending aorta, while pleural fluid will be located posterior. Another common pitfall is failing to assess the posterior pericardium for fluid. In the supine patient, free fluid will preferentially relocate posteriorly due to gravity and one may miss this fluid if only visualizing the anterior pericardium. Additionally, clotted blood may appear echogenic and should not be overlooked when interrogating the pericardial spaces.

Finally, it is important to be aware that the FAST exams are dynamic and only valid during the time they are being performed. Any changes in vital signs or symptoms should prompt a repeat FAST examination. Additionally, not all injuries that require emergent laparotomy will produce sufficient free fluid to result in a positive FAST exam—it is still important to utilize good clinical judgment in these patients.

## KEY POINTS

- Make sure to scan completely through Morison's pouch to avoid

missing small areas of free fluid.

- Differentiate intraperitoneal fluid from perinephric fluid by identifying the hyperechoic Gerota fascia.
- Be sure to view both the splenorenal and infradiaphragmatic areas when assessing the LUQ.
- Consider instilling water into the bladder to improve the suprapubic view.
- Remember to repeat the FAST exam with any significant clinical changes.

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## **CLOSING THE BOOK: USING A BEDSHEET TO STABILIZE PELVIC FRACTURES**

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**MICHAEL GOTTLIEB, MD, RDMS AND STUART SWADRON, MD, FRCPC**

Pelvic fractures are most commonly caused by motor vehicle collisions and falls from height. They are associated with a significant risk of morbidity and mortality. There are three main patterns: lateral compression (most common), anteroposterior compression, and vertical shear. All of these may result in significant and life-threatening pelvic hemorrhage. Most bleeding in pelvic trauma is due to venous injury, but arterial injury may be identified in up to 15% of patients. Retroperitoneal bleeding from pelvic trauma can be severe; up to 4 L of blood can enter this potential space.

Once a pelvic fracture is identified, the pelvis should be stabilized to reduce further bleeding. The ideal treatment is external or internal fixation along with angiographic embolization in the case of arterial bleeding. Studies have suggested that it is preferable to perform angiography prior to external fixation in unstable patients with pelvic fractures. However, in the initial resuscitation, other clinical priorities (e.g., airway, breathing) may overshadow the identification and treatment of a potential pelvic fracture. While the patient is being evaluated and treated for concomitant injuries, the pelvis should be preliminarily stabilized. This is to prevent further shear injury to pelvic vessels, not necessarily to tamponade any active bleeding as previously thought.

There are a number of commercial devices available to stabilize the pelvis while awaiting definitive care. However, if any of these devices are

not immediately available, a bedsheet may also be utilized for this purpose. The bedsheet should be folded lengthwise and then wrapped around the patient's hips with the force directed inward on the greater trochanters. A common pitfall with this technique is wrapping the bedsheet over the iliac crests, which may actually worsen pelvic bleeding. Once the bedsheet is wrapped around the patient's pelvis, it should be pulled crosswise until it is snug (but not so tight as to pull the sides of the pelvis closer together). This may be augmented by taping the lower extremities in internal rotation. Towel clips or hemostats may also be used to hold the bedsheet in position during resuscitation.

One particularly high-risk event during the resuscitation of a multiply injured trauma patient with a pelvic fracture is rapid sequence intubation (RSI). Clinicians may underestimate the degree of associated hemorrhagic shock, resulting in a profound and exaggerated drop in blood pressure after the administration of an induction agent. Furthermore, the muscle relaxation associated with the paralytic drug component of RSI may cause an acceleration of hemorrhage due to a loss of muscular tone across an unstable fracture site. Thus, when pelvic fracture is suspected on primary survey, it is recommended that some sort of stabilization is in place prior to the administration of the RSI agents. A drop in blood pressure should be anticipated and mitigated by administration of fluids and/or blood products as appropriate. Once other major injuries have been addressed, the patient should be admitted or transferred for definitive care.

## KEY POINTS

- All major classes of pelvic fracture (with the exception of avulsion injuries) can cause life-threatening hemorrhage.
- In multiply injured patients with suspected pelvic fracture, stabilize the pelvis prior to rapid sequence intubation.
- Pelvic binders are fastened snugly over the greater trochanters, not across the iliac crests.
- A bedsheet secured with towel clips or hemostats can be used in the place of a commercially applied device.

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## IS SPINAL IMMOBILIZATION STILL NECESSARY?

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**JOSEPH PALTER, MD**

Spinal injuries remain a significant source of morbidity and health care cost for patients across the United States. The incidence of trauma-related spinal column injuries is estimated at 30,000 per year, with roughly one-third representing acute spinal cord injuries. These data notwithstanding, 1 to 5 million patients are transported via emergency medical services (EMS) in spinal immobilization with cervical collar and long rigid board, indicating an overzealous use of spinal precautions.

Spinal immobilization has been a hallmark of EMS prehospital care since the 1970s. Research in the decades following this implementation showed that there was a 31% decrease in patients arriving with complete spinal lesions. Though initially attributed to the use of spinal immobilization, several other confounding factors (improved EMS infrastructure and improved vehicle safety) likely also played a role. To date, no rigorous studies have demonstrated that spinal immobilization reduces further injury, nor improves short-or long-term neurologic outcomes. The U.S. Consortium of Metropolitan Medical Directors Position Statement even acknowledges that current EMS protocols are “based principally on historical precedent, dogma and medicolegal concerns, and not on scientific evidence.”

Spinal immobilization is not a benign procedure, and the potential for hazard exists. Ironically, those patients whom spinal immobilization looks to protect from further injury may actually be those most at risk for iatrogenic morbidity. In patients with incomplete cervical spinal cord injury, respiratory abilities already may be compromised and fixed supine position further restricts respiratory function and increases risk for asphyxiation. Elderly patients and those with para-or quadriplegia (from new spinal cord injury)

are unable to readjust effectively on the rigid long board, increasing their risk for the development of pressure sores and tissue necrosis, even in short periods of time. Many studies have documented an adverse role of the cervical collar in patients with increased intracranial pressure and in rare cases there can even be distraction of unstable fractures. Finally, the pain and discomfort that spinal immobilization adds to the management of the trauma patient should not be underestimated—it is often the most uncomfortable part of the entire process for many patients.

Multiple studies have demonstrated that spinal immobilization is unnecessary in the penetrating trauma patient, even in the setting of suspected spinal column involvement. Haut et al. reviewed over 30,000 cases and concluded that to potentially benefit one penetrating trauma patient, >1,000 patients need to be immobilized. In contrast, the same study deduced that to potentially contribute to one patient's harm or death, only 66 need to be immobilized. In short, for spinal immobilization in penetrating trauma, that's a number needed to treat (NNT) of 1,000 and a number needed to harm (NNH) of 66.

The window of patients with potential for theoretical benefit from spinal immobilization is especially narrow; they are blunt trauma patients with unstable spinal column injuries but intact or incompletely injured spinal cords. Some authors have reasoned that given the considerable force required to fracture the spine, it is the initial impact that most often causes injuries, not subsequent extraction or transport.

Hauswald et al. compared two University Hospitals, one with an EMS system (Albuquerque, New Mexico) and one without (Kuala Lumpur, Malaysia), and examined the effect of spinal immobilization on trauma patients. Over a 5-year period, none of the 120 patients with spinal injury seen at the University of Malaya were immobilized during transport, whereas all 334 patients seen at the University of New Mexico were. They concluded that there was a <2% chance that immobilization had any beneficial effect, though their conclusion may be confounded by differing hospital populations. Despite this small study, certain patients may still benefit from some form of spinal immobilization to prevent further injury, but that exact population has yet to be fully defined.

With the advent and success of clinical criteria to reduce cervical spine imaging in emergency departments (EDs), some EMS systems have looked to extrapolate these protocols out to the prehospital setting. Early studies displayed good intraevaluator reliability between EMS personnel and ED physicians in the use of clinical criteria to remove cervical collars. Dormier et al. prospectively evaluated such a modified clinical criteria with EMS

evaluations in the prehospital setting and found they were able to reduce patients requiring spinal immobilization by 39% while maintaining high sensitivity for injury.

While spinal immobilization certainly still has its place in the setting of blunt trauma patients with suspected spinal injuries, the ubiquitous application of such immobilization contributes to worse outcomes without clear benefit, especially in low-risk patients. More research is needed to define a clear population that benefits from spinal immobilization.

## KEY POINTS

- Spinal immobilization may be protective of the injured spine during patient transport in some patients, although evidence for its efficacy is lacking.
- Spinal immobilization carries significant morbidity and may even result in life-threatening complications such as airway compromise and increased intracranial pressure.
- Spinal immobilization is not necessary for patients with penetrating trauma.
- Although evidence does not support routine spinal immobilization in blunt trauma, medicolegal concerns and other cultural factors continue to drive the widespread practice.

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## ARE VITAL SIGNS RELIABLE AT ASSESSING DEGREE OF HEMORRHAGE?

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**MICHAEL K. SAFA, MD**

Hemorrhage is defined as the escape of blood from a ruptured vessel. It can be divided into traumatic and nontraumatic causes. Common traumatic injuries leading to significant hemorrhage include solid organ injury, long bone fractures, or vascular injury. Unrecognized hemorrhagic shock is the most common cause of preventable death following traumatic injury. Common nontraumatic sources of hemorrhage include gastrointestinal bleeding, aneurysmal bleeding, or ruptured ectopic pregnancy. Initial evaluation of a patient in the emergency department (ED) with hemorrhage involves discerning the degree of blood loss the patient has sustained. Early detection of hypovolemia in an acutely bleeding patient is critical to prompt treatment of hemorrhage, including replacing lost blood.

Typically, an adult patient's circulating blood volume is 7% of ideal body weight or about 5 L for a 70-kg individual. Clinicians are generally taught how to approximate blood loss based on vital signs (heart rate, blood pressure, and respiratory rate). We often expect that heart rate and respiratory rate will increase while blood pressure decreases in the setting of hemorrhage. Our dependence upon vital signs may be a function of the broad participation in the Advanced Trauma Life Support (ATLS) course of the American College of Surgeons. ATLS classifies degree of hypovolemic shock secondary to hemorrhage into four groups and is firmly rooted in the analysis of the vital signs (see [Table 276.1](#)).

**TABLE 276.1 ATLS CLASSIFICATION OF SHOCK****CLASS OF HEMORRHAGIC SHOCK**

|                                       | I         | II        | III         | IV        |
|---------------------------------------|-----------|-----------|-------------|-----------|
| Blood loss (mL)                       | Up to 750 | 750–1,500 | 1,500–2,000 | >2,000    |
| Blood loss (% blood volume)           | Up to 15  | 15–30     | 30–40       | >40       |
| Pulse rate (beats per minute)         | <100      | 100–120   | 120–140     | >140      |
| Blood pressure (mm Hg)                | Normal    | Normal    | Decreased   | Decreased |
| Respiratory rate (breaths per minute) | 14–20     | 20–30     | 30–40       | >40       |

Source: Fildes J, et al. *Advanced Trauma Life Support Student Course Manual*. 8th ed. American College of Surgeons, 2008.

Unfortunately, the ATLS classification of hemorrhagic shock has never been supported by prospective evidence. One very large observational study demonstrated that while relationships between the derangement of vital signs and amount of blood lost do exist, these are not to the degree and consistency suggested by the ATLS classification. For example, in this study, the median systolic blood pressures in patients who would be considered to be in hemorrhagic shock classes III or IV based on heart rate were not decreased (133 mm Hg and 130 mm Hg, respectively). Another large observational study found similar results. For example, in this study, it was observed that patients with an estimated blood loss of >40% had an average systolic blood pressure of 120 mm Hg. In fact, in cases of traumatic hemorrhage, the ATLS classification overestimates both the decrease in blood pressure and the increase in respiratory rate that accompanies tachycardia. Ultimately, the proposed pathophysiologic relationships do not seem to reflect what actually happens clinically.

In clinical practice, patients may have subtle changes in their heart rate and blood pressure in the setting of hemorrhage but may go unrecognized as they are not likely to be as dramatic as providers are led to believe in ATLS training. Delay in recognition of life-threatening bleeding and its treatment results.

There are many factors that may make vital signs less likely to correlate with degree of hemorrhage. These include (but are not limited to) patient age, pain severity, type of injury, and medications. Children and young healthy patients, for example, may demonstrate a significant physiologic reserve to bleeding. An older patient or a patient taking beta-blockers will have a blunted tachycardic response. A patient with traumatic intra-abdominal hemorrhage or hemorrhage secondary to ruptured ectopic pregnancy may

prop up his or her blood pressure with increased vagal tone in response to hemoperitoneum. In subacute or slow bleeding, patients may not have vital sign changes secondary to physiologic compensation. Emergency physicians encounter this frequently in patients with chronic gastrointestinal bleed in whom the normal vital signs belie the severe drop in hemoglobin.

Ultimately, using vital signs for determining the amount of blood loss in either traumatic or nontraumatic hemorrhage is helpful but unreliable. Knowing some of the key caveats above will help reduce our overreliance on vital signs and the resulting complacency that can result in a delay in recognizing ongoing blood loss.

## KEY POINTS

- Vital signs changes are inaccurate in estimating blood loss in acute bleeding.
- The elderly, patients on nodal blocking medications, and those with intra-abdominal hemorrhage may present without tachycardia in severe acute hemorrhage.

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## THE ABCs OF MAJOR BURNS

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**MARY L. CHEFFERS, MD AND STUART SWADRON, MD,  
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The American Burn Association defines major (severe) burns as 25% total body surface area (TBSA) or greater in patients aged 10 to 40 years (excluding superficial burns), 20% TBSA or greater in children under 10 years and adults over 40 years of age, 10% TBSA or greater full-thickness burns, or any burn that involves the eyes, ears, face, hands, feet, or perineum that is likely to result in cosmetic or functional impairment. The majority of major burn patients will be brought to the nearest emergency department (ED) by Emergency Medical Service (EMS) providers given the possible need for airway management. For emergency physicians, management of major burns should be focused on the essentials: securing the airway, appropriate fluid resuscitation, identifying concomitant injury, stopping any continuing burn, treating pain, and obtaining definitive care. The common pitfalls where providers deliver suboptimal care fall into three categories: failing to secure the airway, inadequate fluid resuscitation, and failing to promptly transfer the patient to burn units.

### **AIRWAY**

Prompt intubation for protection of the airway should be performed in anyone with suspected inhalation injury, both by mechanism and by physical findings. These include burns suffered in enclosed spaces, long exposure time, singed nose hairs, and soot in the nasopharynx or oropharynx. Airways can close quickly with fluid resuscitation as tissue injury causes capillary leak phenomenon. Intubation should be seriously considered in any patient who has >50% TBSA burns. This is due to the fact that they often require large fluid volumes, have severe systemic inflammatory response syndrome

(SIRS) reactions, and require large doses of opioid analgesia. Additionally, intubation should be considered when a burn victim needs to be transported a long distance. Once intubated, ventilation strategy should follow the National Institutes of Health ARDSNet protocol to avoid both barotrauma and atelectrauma.

## FLUID RESUSCITATION

This is the intervention that most affects mortality associated with major burns. Both under- and overresuscitation are associated with poor patient outcomes. In the last several years, overresuscitation (“fluid creep”) has been the most common error seen at burn centers. Overresuscitation leads to complications that include prolonged ventilator time and associated morbidity; congestive heart failure; compartment syndrome of the abdomen, globe, and extremities; and extension of the zone of coagulation. Underresuscitation can also lead to extension of the zone of coagulation in addition to hypovolemic and distributive shock, which are difficult to reverse. A simple solution to the problem of appropriate fluid resuscitation is to correctly apply the clinical tools available to calculate %TBSA of burn and then calculate the resuscitation volume needed using the Parkland or other validated formulas. Most reviews performed by burn centers suggest that the error lies in the calculation of %TBSA affected and not in the subsequent fluid requirement calculation.

No clinical tool has been shown to be superior to others in estimating burn size. The three most common for adults include the “Rule of 9’s,” the “Palm-1%,” and the Lund-Browder chart. There are some common pearls that arise in applying these rules that are worth mentioning:

- Only partial-thickness burns and worse should be counted, and the zone of stasis or hyperemia surrounding the zone of injury should not be included. This can be difficult with scald and contact burns, as initially it may be hard to see their boundaries.
- The area should be cleaned to distinguish between soiled areas bordering a wound and burn eschar.
- When noncontiguous burns are present, using the palm as 1% estimate may be more accurate.
- There is a modified clinical tool to estimate %TBSA in children.

Overestimating the size of the burn may at times be purposeful or subconscious. Providers’ inclination may be to think that underestimating the size of the burn may be more harmful than overestimating as patients may

not meet transfer criteria for a burn unit. Interestingly, the data support this theory in that the burns most likely to be overestimated are those that are close to transfer criteria for partial-thickness burns (10% to 20% TBSA).

Once the %TBSA burned is calculated and the patient's weight is estimated, applying a fluid resuscitation formula such as the Parkland formula is fairly straightforward. Remember that all the validated formulas start the clock at the moment of injury, not the time of arrival to the ED. All formulas suggest lactated Ringer's as the fluid of choice. Children should receive dextrose in addition to lactated Ringer's, usually in a separate preparation and run at a different rate. Example: Using the Parkland formula, for an 80-kg patient, 20% TBSA burn means 6.4 L in the first 24 hours. In the first 8 hours, 3.2 L should be given, or 400 mL/hour. If the patient arrives 2 hours after the injury, those 3.2 L need to be given in 6 hours, at a rate of close to 500 mL/hour. Another common error in fluid resuscitation is forgetting to reduce the rate of fluids after the 8-hour mark. In the example above, 3.2 L should be given over the remaining 16 hours, which means that fluids should be reduced to 200 mL/hour after the 8-hour mark postinjury.

## TRANSFER

The final pitfall in managing major burn patients is inappropriate or delayed transfer to burn centers. The American Burn Association provides transfer criteria that should be reviewed periodically. The threshold is low for transfer of these patients as care for burns involves multidisciplinary attention. Delaying transfer can cause serious harm to these patients, and when in doubt, the local burn center can be called for consultation. Any concomitant injury including trauma, CO poisoning, cyanide poisoning, or electrical injury should be addressed prior to transfer in order to allow for safe transfer.

In summary, major burns are rare, but emergency physicians need to have a clear management approach. Focusing on protecting the airway, making accurate estimations of %TBSA of burn, applying fluid resuscitation formulas precisely, and promptly transferring to burn units will avoid the common pitfalls associated with burn management.

### KEY POINTS

- Airway and breathing are always the first consideration in a patient with major burns. Prompt intervention with advanced airway management can be lifesaving.

- Early intubation may also be an important part of treating severe pain and shock.
- Both over- and underresuscitation with fluids continues to be problematic. The greatest source of error appears not to be the formula used, but rather in the estimation of TBSA.
- Early transfer to burn centers is important for patients who meet criteria.

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## **WHEN CAN INTERVENTIONAL RADIOLOGY (IR) BE YOUR FRIEND IN TRAUMA?**

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MBA**

Trauma is the leading cause of death in people under the age of 40 with an estimated 5 million deaths worldwide per World Health Organization (WHO) data. Hemorrhagic shock remains the leading cause of death in trauma patients and accounts for 30% to 40% of trauma mortality. Early management of trauma victims, especially those in hemorrhagic shock, is resuscitation and the identification of life-threatening bleeding to minimize the triad of trauma: acidosis, hypothermia, and coagulopathy. First described in 1972, angiography and angioembolization have become important treatment modalities to stop bleeding in the multiply injured patient. The interventional radiology (IR) procedures may supplement standard surgical intervention by providing hemorrhage control prior to surgery or supplant surgical intervention altogether. Moreover, the nonoperative management of blunt abdominal trauma is becoming increasingly more common. A minimally invasive management strategy circumvents the tissue trauma and anesthesia risks associated with more traditional surgical approaches. The three most common modalities utilized include (1) balloon occlusion—an angioplasty balloon is inflated proximally to a major arterial injury to stabilize the patient for definitive surgical or endovascular repair; (2) transarterial embolization—selected embolic agents including Gelfoam, polyvinyl alcohols, and coils are used for arterial occlusion; and (3) stent grafts—used primarily for large vessel injuries.



# ROLE OF ENDOVASCULAR TREATMENT IN ABDOMINAL TRAUMA

The spleen is the most commonly injured solid organ followed by the liver and the kidney. Splenectomy used to be the standard of care for traumatic splenic injuries, but due to impairments in short-and long-term immunity with some reviews noting a 50% increased risk of postoperative infections after splenectomy, embolization is a viable alternative with the same survival and decreased need for splenectomy. Importantly, embolization does not obliterate the spleen with usually half the splenic bulk and serologic markers of immune function preserved. Currently, the accepted indication for endovascular treatment is the presence of active extravasation or pseudoaneurysm formation in stable patients with splenic injuries. Intravenous contrast-enhanced computed tomography (CT) has been shown to be useful in screening for both pseudoaneurysm formation and extravasation.

Hepatic trauma can involve extensive damage to the hepatic arteries, hepatic veins, and portal veins with the mortality rate of surgery for blunt hepatic trauma reaching >33%. The use of embolization may be the treatment of choice for stable patients, and its use is increasing in unstable patients. Additionally, given the dual blood supply of the liver, the risk of infarction is less. In general, patients who have extensive renal injury (American Association for the Surgery of Trauma grades 4 and 5) usually have other solid organ injuries and are taken to surgery for nephrectomy. Embolization may be a useful adjunct in renal artery injury when used to maximize preservation of viable renal tissue.

Pelvic hemorrhage most commonly originates from fractured bones or disrupted pelvic veins rather than an arterial injury. Many of the complications arise from subsequent hemorrhagic shock and associated organ injury, which can eventually lead to persistent hemorrhage or abdominal compartment syndrome. Current treatment guidelines for unstable pelvic fractures include external fixation with pelvic binders for tamponade, aggressive resuscitation, embolization, and pelvic packing. With the use of CT, accurate identification of arterial extravasation, pseudoaneurysm formation, arterial truncation, or arteriovenous fistula malformation is possible. All of these may be amenable to endovascular therapy.

In addition, IR techniques can be used to stent acute traumatic aortic injury and to control hemorrhage in traumatic extremity injuries with evidence of active extravasation, pseudoaneurysm formation, and arterial occlusion/transection.

In summary, IR is playing an increasingly central role in the management of hemorrhage in the multiply injured patient—it is often the best option.

## KEY POINTS

- IR techniques can be lifesaving in acute hemorrhage and offer advantages over traditional surgical approaches.
- Hemorrhage of solid organs, such as the spleen, liver, and kidney, can be treated with endovascular techniques.
- Hemorrhage from pelvic fractures as well as extremity injuries are also indications.

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# DON'T MISS THE GAMEKEEPER THUMB

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**BRIAN R. SHARP, MD, FACEP**

The ulnar collateral ligament (UCL) and radial collateral ligament (RCL) are the primary stabilizers of the thumb metacarpal (MCP) joint. They attach proximal to the base of the first MCP head and insert on the volar aspect of the proximal phalanx of the thumb. Injury to the UCL is 10 times more common than injury to RCL and is commonly referred to as a “gamekeeper’s thumb.” It can be a significant injury if missed as inadequately treated injuries can lead to chronic instability, persistent pain, decreased pinch strength, or degenerative changes.

Gamekeeper’s thumb or rupture of the UCL was first described by Campbell in 1955. The historical basis of the name is an injury resulting from Scottish gamekeepers twisting the necks of wounded rabbits while hunting. It is now commonly referred to as skier’s thumb as thumb injuries are second only to knee injuries in frequency among skiers. It is also commonly seen in ball sports, falls, and activities of daily living (predominantly falls onto an outstretched hand). The mechanism is typically an abrupt and significant radial stress (valgus force) onto an abducted thumb that forces abduction and hyperextension of the MCP joint. The UCL avulses from proximal phalanx in 90% of cases.

Examination will demonstrate tenderness on the ulnar aspect of the thumb MCP, often with swelling and ecchymosis that can involve the entire joint. Occasionally, a palpable mass is appreciated on the ulnar side (which is the displaced end of the UCL). The injury can result in weak pinch strength and the inability to resist adduction stress. The key physical examination maneuver is to apply a valgus stress to both the affected and unaffected thumbs. This will demonstrate a loss of integrity of the UCL. It is performed

by grasping the MCP joint of the thumb with one hand (stabilizing it from rotation) and the distal thumb with the other hand while applying a valgus stress across the MCP joint. This should be performed in both a neutral position (0 degrees of flexion) as well as flexion (30 degrees). The flexed position allows the volar plate (a very thick ligament that prevents hyperextension) to relax, making the test more sensitive and often detecting an incomplete UCL rupture. A positive test is determined with increased laxity or the lack of an end point (see [Table 279.1](#)). Some surgical texts cite laxity of >30 degrees of the proximal phalanx on the MCP head or >15 to 20 degrees more than the unaffected side, but this can be hard to determine definitively. Making this more difficult, in some individuals, there is some normal discrepancy in joint laxity between thumbs.

**TABLE 279.1 GRADES OF ULNAR COLLATERAL LIGAMENT (UCL) INJURY**

|         |  |
|---------|--|
| Grade 1 | Pain only with valgus stress (no increase of laxity)   |
| Grade 2 | Pain and a mild increase in UCL laxity (often flexed position only)                          |
| Grade 3 | Marked laxity of the UCL, often with no pain on the stressed ligament (complete UCL rupture) |

X-ray is the initial test of choice to evaluate for a commonly associated “gamekeeper’s fracture” or avulsion fracture at the base of the proximal phalanx. There is questionable utility of ultrasound or other advanced imaging modalities.

If a UCL injury is suspected, the patient should be placed in a thumb spica splint with the thumb flexed to ~20 degrees. Referral to a hand surgeon is indicated. Recommendations for period of immobilization vary, but generally include 4 weeks of initial immobilization. With conservative management, the patient will next begin gentle passive range of motion exercises (with immobilization when not performing exercises). Surgery is generally recommended if there is a complete rupture, due to more predictable outcomes, within 3 to 4 weeks of injury. Surgery may be effective for joint stability even if delayed for years after the injury.

## KEY POINTS

- Ulnar collateral injury occurs with abduction and hyperextension of

the thumb.

- You need to perform a UCL stress examination in a neutral position as well as in flexion (30 degrees).
- Initial treatment of a suspected UCL injury is a thumb spica splint and hand surgery referral.

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## ADMIT DISPLACED SUPRACONDYLAR FRACTURES FOR NEUROVASCULAR CHECKS

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**ALLISON S. LUU, MS, MD AND ERIC WEI, MD, MBA**

Supracondylar fractures account for 16% of all pediatric fractures and make up over half of all pediatric elbow fractures. In 70% of patients, these result from an injury caused by a fall on an outstretched arm (FOOSH) in which force is transmitted through the olecranon to a weak supracondylar area resulting in a fracture. These fractures occur most commonly in children between the ages of 3 and 10 with peak incidence between 5 and 7 years of age.

There are two types of supracondylar fractures, those that occur during extension or hyperextension and those that occur during flexion. More than 95% of these fractures are the extension type, those caused by a FOOSH. In the flexion type, the mechanism is usually a fall from height onto a flexed elbow.

In order to diagnose a supracondylar fracture, it is important to remember that there are several things to look for on x-ray. The first is the displacement of fat pads caused by a fracture hemorrhage. The normally present anterior fat pad will turn into a triangular shape known as the “sail” sign. In addition, you may see a posterior fat pad, which is always abnormal regardless of its shape and is diagnostic for a fracture.

It is also important to keep in mind the order at which the ossification centers or growth plates appear and disappear. CRITOE is a well-known mnemonic used to help predict when these structures appear. The ossification centers then fuse over time with the medial epicondyle fusing last ([Table](#)

280.1). A common error is to confuse a medial epicondyle growth plate for a fracture, as it is the last to fuse in a pediatric elbow. If there is doubt, comparing radiographs with the opposite uninjured elbow may be helpful.

**TABLE 280.1 CRITOE MNEMONIC FOR ELBOW OSSIFICATION CENTERS IN CHILDREN**

| OSSIFICATION CENTER           | AGE OSSIFICATION BECOMES VISIBLE (YEARS) | AGE OSSIFICATION FUSES (YEARS) |
|-------------------------------|--|--------------------------------|
| Capitellum                    | 1  | 12                             |
| Radial head                   | 3  | 15                             |
| Internal (medial) epicondyle  | 5  | 17                             |
| Trochlea                      | 7  | 12                             |
| Olecranon                     | 9  | 15                             |
| External (lateral) epicondyle | 11                                       | 12                             |

The last thing to note is that in a normal radiograph, the anterior humeral line should pass through the middle third of the capitellum. If the capitellum is anterior to the anterior humeral line, this is diagnostic for a flexion-type supracondylar fracture. A capitellum that is seen posterior to the anterior humeral line is diagnostic of an extension-type supracondylar fracture. Extension-type supracondylar fractures are separated into the classifications based on their treatment ([Table 280.2](#)).

**TABLE 280.2 CLASSIFICATION OF EXTENSION-TYPE SUPRACONDYLAR FRACTURES**

|          | CHARACTERISTICS   | EMERGENCY DEPARTMENT TREATMENT   |
|----------|---|--|
| Type I   | Minimally or nondisplaced fracture (fat pad abnormality)  | Splint with a posterior long arm in slight flexion and discharge home with close follow-up within 24 h                                   |
| Type II  | Displaced fractures or those with an anterior humeral line that passes anterior to the capitellum on the lateral radiograph, with intact posterior cortex | Pediatric orthopedist evaluation with choice of closed reduction vs. percutaneous pinning. Hospital admission for neurovascular checks   |
| Type III | Significant displacement of the distal humerus with disruption of the posterior cortex  | Pediatric orthopedist evaluation with closed or open reduction, percutaneous pin placement, and hospitalization for neurovascular checks |

Regardless of the type of fracture, the elbow should initially be splinted in a position of comfort at ~20 to 30 degrees of flexion and elevated above the level of the heart. Care should be taken not to splint the limb in full extension as this could damage the neurovascular bundle, especially in displaced or unstable fractures. In addition, splinting at >90 degrees of flexion greatly increases the forearm compartment pressures, putting the patient at risk for compartment syndrome.

According to current literature, neurovascular injuries occur in 12% of all supracondylar fractures. The rate increases with the degree of displacement. In extension-type fractures, the median nerve is injured most commonly, specifically the anterior interosseous branch (28% to 60%). The integrity of the anterior interosseous nerve can be tested using the strength of the patient while holding the “OK” sign using the thumb and index fingers. The second most commonly injured nerve is the radial (26% to 61%), followed by the ulnar (11% to 15%). In flexion-type supracondylar fractures, the ulnar nerve is the most commonly injured. This can be tested by examining the strength of the intrinsic hand muscles. Motor impairment at the time of injury is almost always caused by neuropraxia and usually improves within 3 months. Further workup is not needed unless the deficit persists beyond this period.

Compartment syndrome should be suspected in a patient with increasing pain medication requirements or if paresthesias develop. This is an emergency; the patient should be brought to the operating room for fasciotomy. The feared complication of compartment syndrome is Volkmann’s ischemic contracture. Volkmann contracture results from an untreated compartment syndrome of the forearm and is characterized by fixed flexion at the wrist and elbow, with the forearm in pronation and the metacarpal-phalangeal joints in extension.

Due to the high incidence of neurovascular injury and the feared permanent consequences of Volkmann ischemic contracture, most type II and all type III supracondylar fractures should be admitted for neurovascular checks with urgent or emergent pediatric orthopedic consult for possible operative repair.

## KEY POINTS

- Supracondylar fractures with significant displacement are at high risk for neurovascular complications and poor outcomes. Admission and observation for significantly displaced fractures is indicated.
- Compartment syndrome should be suspected in a patient with



increasing pain medication requirements or if paresthesias develop.

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# **KNOW THE RADIOGRAPHIC SIGNS OF SCAPHOLUNATE DISLOCATION**

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**NICHOLAS ABRAHAM, MD AND STUART SWADRON, MD,  
FRCPC**

Scapholunate dislocation or dissociation (SLD) is widely recognized as the most common ligamentous injury of the wrist. The injury occurs in isolation as well as with a variety of fracture-dislocation patterns, including up to 30% of intra-articular distal radius or carpal fractures. Mechanism of injury is most commonly a direct impact force to the hand and wrist over the thenar eminence with the wrist positioned in extension, ulnar deviation, and carpal supination. This results in an acute tear of the scapholunate ligament creating a gap between the lunate and the proximal pole of the scaphoid. Additionally, the injury can coexist with a rupture of the radioscapholunate ligament resulting in palmar rotation (or rotary subluxation) of the scaphoid.

SLD is frequently missed on initial presentation and can be very subtle, especially when in isolation or associated with other more severe injuries. It is inherently more difficult to detect because it is often present without a fracture. Clinically, patients present with varying degrees of grip weakness, limited motion, dorsoradial swelling, and point tenderness. These symptoms and signs are often associated with a clunking or snapping sensation with wrist movement. The scaphoid tilt test is valuable to determine the presence of injury. A positive test has been described as diagnostic when performed by an experienced clinician.

## **SCAPHOID TILT TEST**

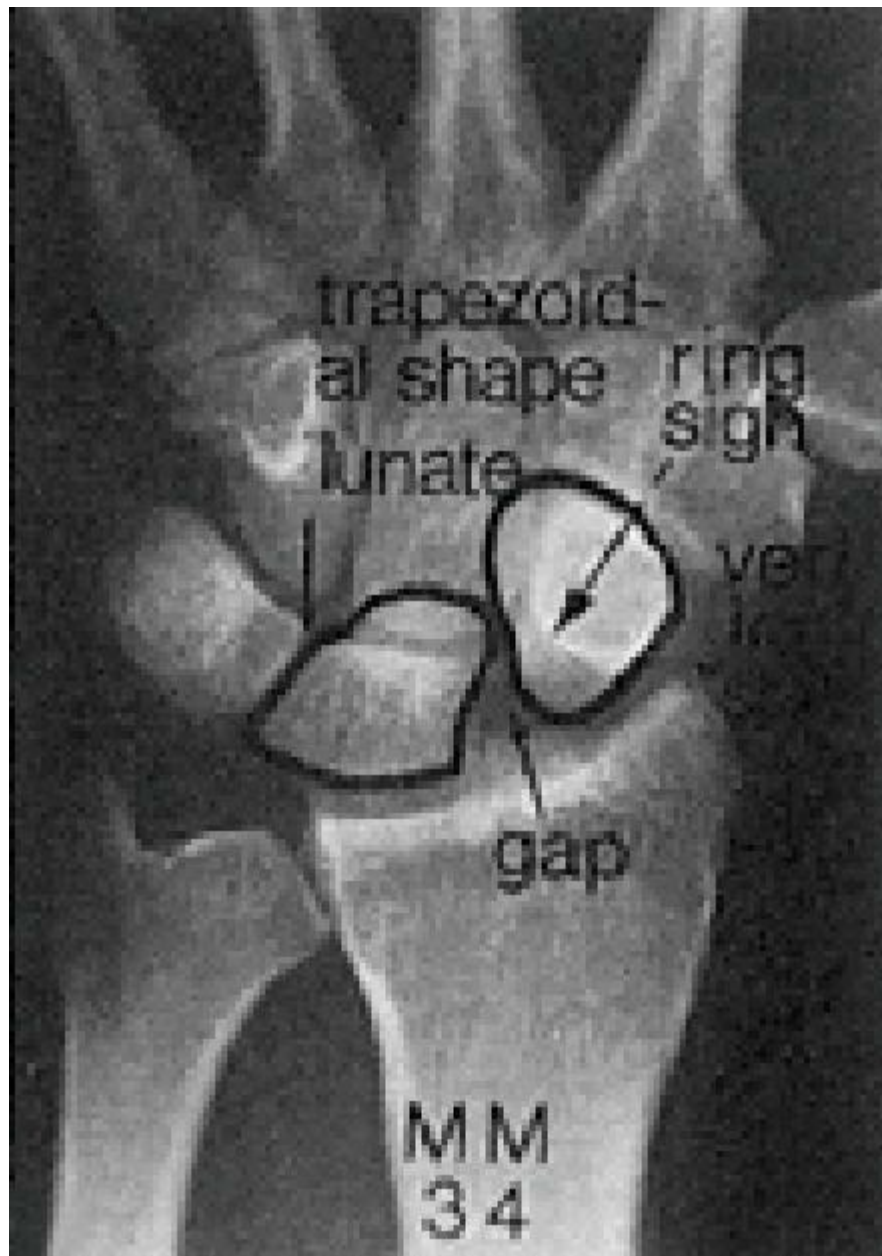
- 1) Place four fingers behind the radius with your thumb on the tuberosity

of the scaphoid.

- 2) Use your other hand to passively move from ulnar to radial deviation.

When the hand is ulnar deviated, the scaphoid is in an extended position in line with the forearm, while in radial deviation, the scaphoid is flexed. Applying pressure to the tuberosity while moving the hand from ulnar to radial deviation prevents the scaphoid from flexing. If the scapholunate ligaments are disrupted, the proximal pole moves dorsally out of alignment with the radius, often inducing pain on the dorsoradial aspect of the wrist. When the applied pressure is released, the scaphoid self-reduces over the dorsal rim of the radius inducing the typical clunking or snapping as described above.

Once suspected, the diagnosis is confirmed by the presence of one or more features seen on radiographs. Emergency physicians should know the carpal bones and have a standardized routine for evaluating wrist films. While many mnemonics exist, one example is “So Long To Pinky, Here Comes The Thumb” for the scaphoid, lunate, triquetrum, pisiform, hamate, capitate, trapezoid, and trapezium, respectively. On the anteroposterior (AP) view, the clinician should look for widening of the scapholunate joint space. Examining this space should be a routine part of a clinician’s wrist film reviewing process. A measurement of  $>3$  mm is pathognomonic, named the “Terry Thomas” sign after the popular 1960s comedian with a trademark gap in his front teeth. More recently, this gap has been dubbed the “David Letterman” sign after the more contemporary comedian. The second sign to be aware of is the cortical ring sign, formed by a foreshortened scaphoid, due to rotary subluxation and volar tilt, causing a visible ring-shaped double density at its distal pole (see [Figure 281.1](#)). The lateral view should also be closely examined to ensure that the radius, lunate, and capitate form a straight line. The proximal portion of the scaphoid is projected over the lunate with the distal segment volar. A line drawn through the center of the lunate and scaphoid makes up the scapholunate angle, which should be between 30 and 60 degrees. Make sure to also closely examine the radiographs for perilunate or lunate dislocations as well as distal radius, radial styloid, or scaphoid fractures since they are closely associated with SLD.



**Figure 281.1** AP radiograph of scapholunate dislocation. (From Bucholz RW, Heckman JD, eds. *Rockwood and Green's Fractures in Adults*. 5th ed. Philadelphia, PA: Wolters Kluwer, 2002.)

Patients with scapholunate dislocation should be placed in a thumb spica splint with the wrist in a neutral position or 10 to 15 degrees of dorsiflexion. Urgent referral to an orthopedist or hand surgeon for surgical repair is required as these injuries are typically difficult to repair with unpredictable results. The most common modalities for repair are closed reduction with percutaneous pinning or open reduction and internal ligamentous repair. Prompt repair during the acute phase of the injury carries the highest potential for satisfactory outcomes. We must catch this diagnosis early to prevent associated sequelae including severe and often debilitating

degenerative arthritis.

## KEY POINTS

- Scapholunate dislocation or dissociation (SLD) is the most common ligamentous injury of the wrist.
- Key x-ray findings include a widening of the scapholunate joint space and the cortical ring sign.
- The lateral x-ray is important to check for associated lunate and perilunate dislocation.

## SUGGESTED READINGS

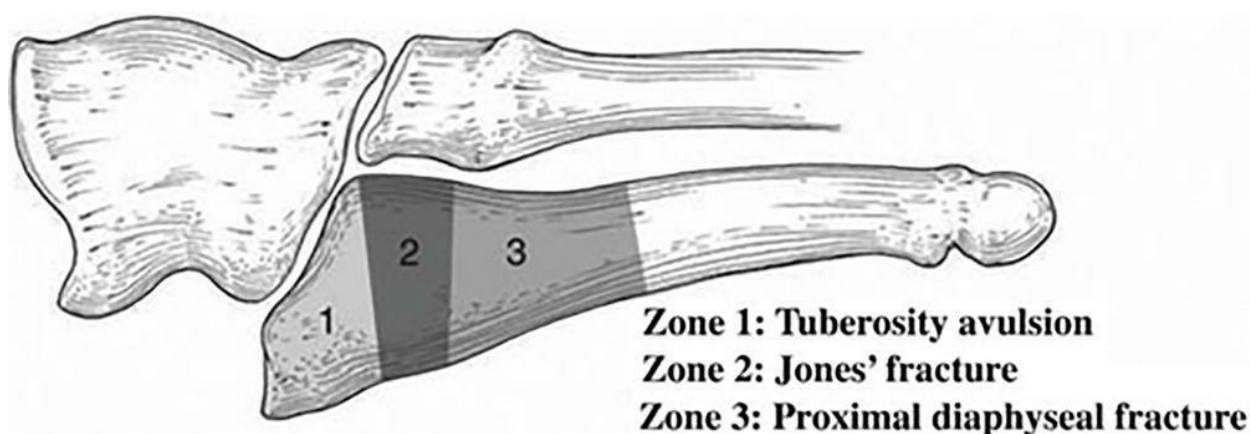
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# KNOW THE DIFFERENCE BETWEEN JONES AND PSEUDO-JONES FRACTURES

**BRIAN R. SHARP, MD, FACEP**

The proximal fifth metatarsal is the most common site of midfoot fractures and accounts for 45% to 70% of all metatarsal fractures. The best known is perhaps the Jones fracture, which was first described in 1902 by Sir Robert Jones after he injured his foot dancing. However, this fracture is actually far less common than the pseudo-Jones or fifth metatarsal avulsion fracture.

The fifth metatarsal is composed of three anatomic zones, each with a corresponding fracture. Significant differences in prognosis and treatment can depend on mere millimeters, making it very important to be able to differentiate the three zones and corresponding fracture types (see [Figure 282.1](#)).



**Figure 282.1** Fifth metatarsal fractures. (Modified from Lawrence SJ, Botte MJ. Jones' fractures and related fractures of the proximal fifth

metatarsal. *Foot Ankle*. 1993;14(6), Figure 3, page 360, with permission.)

## **ZONE 1 TUBEROSITY AVULSION FRACTURES: PSEUDO-JONES/DANCER'S FRACTURE**

Tuberosity avulsion fractures of the fifth metatarsal are often referred to as a pseudo-Jones or dancer's fractures. They represent 90% of fractures at the base of the fifth metatarsal. Although typically visible on standard anteroposterior (AP), lateral, and oblique foot radiographs, ankle films are often needed to visualize the fracture—up to 23% are missed with foot radiographs alone. The fracture itself can have a transverse or oblique appearance and always occurs proximal to the intermetatarsal joint between the fourth and fifth metatarsals. Although an avulsion fracture can involve the metatarsocuboid articulation, it should never involve the intermetatarsal joint between the fourth and fifth metatarsals.

The typical mechanism of an avulsion tuberosity fracture is forced inversion of the foot and ankle while in plantar flexion (e.g., a basketball player landing awkwardly after a jump or runner inverting the ankle on an uneven surface). Tension generated by the peroneus brevis tendon and/or lateral cord of plantar aponeurosis (fascia) avulses the tuberosity. Patients often present complaining of a “sprained ankle” due to the mechanism and relatively mild symptoms.

Treatment is symptomatic and includes at least 3 weeks of a hard-soled or cast shoe ( $\pm$  compression dressing) with a goal of preventing significant plantar flexion and weight-bearing as tolerated. A posterior splint with crutches or a short leg walking cast for 2 to 3 weeks can be used if there is severe pain. Orthopedic referral is indicated if there is  $>3$  mm of displacement, a step-off of more than 1 to 2 mm on the articular surface of the cuboid, or symptomatic nonunion. All of these may ultimately require operative intervention.

Multiple metaphyseal blood vessels and branches of the nutrient artery supply the tuberosity of the fifth metatarsal. Prognosis for these fractures is thus excellent with most patients being asymptomatic at 3 weeks; radiographic union is typically seen at 8 weeks. Complications are unusual but include nonunion or prolonged discomfort. These are more common if there is a step-off on the articular surface and in older patients.

## **ZONE 2 METAPHYSIS/DIAPHYSIS—JONES FRACTURE**

The Jones fracture is an acute fracture of the junction of the diaphysis and metaphysis of the fifth metatarsal—this is where the widened part of the bone begins to thin out as it becomes the shaft of the bone (typically within 1.5 cm of the metatarsal tuberosity) and extends toward the intermetatarsal joint (typically between the fourth and fifth metatarsals). Jones fractures are frequently accompanied by phalanx fractures. There is particular clinical significance to the location of this fracture because it can disrupt the blood supply to the distal portion of the proximal fragment, which is a tenuous “watershed” area.

The mechanism of a Jones fracture is typically a sudden change in direction with the heel off of the ground. This creates either a vertical or lateral force on the forefoot. It is often reported in sports: basketball, soccer, football, and occasionally tennis. A patient will typically present with pain and tenderness in the lateral foot. Jones fractures are thought to be more common in people with a high arched foot shape—this results in increased loading on the lateral foot.

Initial treatment includes ice, elevation, and immobilization in a posterior short leg splint with strict non-weight-bearing status and orthopedic follow-up within 3 to 5 days. Definitive treatment is typically a short leg, non-weight-bearing cast for 6 to 8 weeks.

Prognosis is guarded because of a high incidence of delayed healing and nonunion due to the aforementioned poor blood supply to this region. Even with immobilization, up to one-half later require surgery due to nonunion or refracture. Early surgical intervention with intramedullary screw fixation is becoming increasingly common with a high reported rate of primary union.

## **ZONE 3 PROXIMAL DIAPHYSIS—STRESS FRACTURES**

Diaphyseal stress fractures are seen distal to the ligamentous attachments of the bone (1.5 cm into the diaphysis). These fractures are typically symptomatic for several days prior to presentation and are not an acute injury.

The treatment is immobilization and non-weight bearing for 6 to 10 weeks (similar to that for a Jones fractures), but the prognosis for fracture union is even worse than with Jones fractures. These often require up to 20



weeks of immobilization—early surgery is thus an option.

## KEY POINTS

- An avulsion fracture of the fifth metatarsal occurs with an inversion injury, whereas a Jones fracture is a lateral force while the heel is off of the ground.
- An avulsion fracture of the fifth metatarsal occurs proximal to the intermetatarsal joint between the fourth and fifth metatarsals.
- While an avulsion fracture can be treated with a postoperative shoe, both Jones fractures and fifth metatarsal stress fractures require prolonged immobilization and strict non-weight-bearing status.

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## SEARCH FOR OTHER INJURIES IN PATIENTS WITH SCAPULAR FRACTURE

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**JOHN W. MARTEL, MD, PhD, FACEP**

Scapula fractures are uncommon, with an annual incidence of 10 to 12 per 100,000 people. They typically are associated with high-energy, blunt force mechanisms, including motor vehicle collisions and falls from height, and therefore are commonly associated with other serious injuries. Although they constitute <1% of all fractures, scapula fractures are associated with a 10% to 15% mortality rate. Of critical importance, over 90% of patients have concomitant injuries whose diagnoses may be delayed or overlooked entirely if not carefully considered. In particular, there is risk of coexisting multisystem thoracic, orthopedic, intracranial, intra-abdominal, and neurovascular injury. For example, there is a very high reported incidence (75% to 98%) of associated injuries to the ipsilateral lung, chest wall, and shoulder girdle.

During physical examination, fully conscious patients commonly maintain the affected shoulder in a position of adduction with the extremity itself held close to the chest wall. In the appropriate clinical context, a variety of shoulder findings, including ipsilateral ecchymosis, hematoma, focal tenderness, or crepitus, should raise suspicion for scapula fracture as well as its associated serious injuries. Scapula fractures occur primarily in the body, followed by the neck, glenoid, and acromion in descending order. Three-view shoulder radiographs (anteroposterior [AP], transscapular lateral, and axillary lateral views) aid in rapid diagnosis via evaluation of both the glenohumeral structures and scapular body. In some cases, scapula fractures may also be appreciated on chest x-ray, but they can also be easily missed on

a portable chest film in a critically injured patient. Computed tomography (CT) more effectively delineates the fracture and is also most useful in ruling out associated injury in the stable, multiply injured patient.

With regard to injury patterns, the most common are rib fractures and acute intrathoracic pathology, such as pneumothorax, hemothorax, and lung contusions. These are seen together with scapula fracture (in some combination) in up to two-thirds of cases. In addition, skull fracture and intracranial injuries, including intracranial hemorrhage and cerebral contusion, occur in up to 40% of patients. Lastly, up to 10% of cases are associated with injury to regional vasculature, including the brachial, subclavian, and axillary arteries.

It is also prudent to look for other orthopedic injuries, with special focus on the axial skeleton, pelvis, and extremities. A 10-year retrospective review of blunt trauma admissions at two large, urban level 1 trauma centers reported that patients with scapular fractures tended to have more severe injury severity scores and underlying thoracic injuries. Another retrospective review of the National Trauma Database reported that concomitant injuries to the thorax, upper extremities, and pelvis were associated with greater frequency in patients with scapula fracture.

Fortunately, isolated scapula fractures are generally not associated with permanent disability. The vast majority of cases are managed nonoperatively with application of a sling and early range of motion, with the exception being unstable fractures that require operative intervention.

## KEY POINTS

- Given the high-energy mechanisms that typically break the scapula, a careful trauma evaluation is required in order to avoid missing potentially life-threatening injuries.
- The most common associated injuries are rib fractures, pneumothorax, hemothorax, and lung contusions.

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# Do You Know How to Do ABIs?

JOHN C. RAY, MD

The ankle-brachial index (ABI) is a key diagnostic tool in the assessment of arterial flow in the lower extremities. It is a noninvasive screening tool for the identification of peripheral arterial disease and arterial injury which is used in all clinical settings, including outpatient, inpatient, and the emergency department (ED). The ABI is quickly performed and simple to interpret, and it can aid in the management of patients who present with any concern for lower extremity arterial compromise. Although most frequently utilized in the outpatient setting to quantify the extent of peripheral arterial disease, several ED applications exist (see [Table 284.1](#)).

## TABLE 284.1 INDICATIONS FOR USE OF ANKLE BRACHIAL INDEX IN THE EMERGENCY DEPARTMENT

- Wound infection or cellulitis in an ischemic limb
- Evaluation of foot ulcers or gangrene
- Evaluation of arterial flow in lower extremity traumatic injuries
- Acute limb ischemia associated with a thrombus
- Postsurgical evaluation after angioplasty, stenting, or lower extremity bypass surgery

Simply stated, an ABI is performed by measuring blood pressure in the upper and lower extremities and comparing their respective values. The necessary equipment for performing an ABI is found at the bedside in any standard ED examination room. These include blood pressure cuffs in a variety of sizes, a manual sphygmomanometer, a portable Doppler ultrasound, and ultrasound

gel or lubricating jelly. The following steps delineate the proper technique to measure and calculate ABIs:

- Step 1: Place the patient in supine position.
- Step 2: Choose the appropriate blood pressure cuff size for the patient's upper arm as well as for the patient's ankle, and apply the pressure cuff to the appropriate location. Go one inch above the antecubital fossa for brachial measurements and 2 to 3 inches above the malleoli for ankle measurements.
- Step 3: For *brachial measurements*, apply an adequate amount of gel to the patient's antecubital fossa, and position the Doppler probe at a 45 to 60-degree angle directed toward the patient's head to obtain the clearest arterial pulse signal and keep the probe steadily positioned.
- Step 4: Inflate the brachial blood pressure cuff to 20 mm Hg above the point where the arterial pulse signal is no longer appreciated.
- Step 5: Slowly deflate the brachial blood pressure cuff, keeping the Doppler probe in place, until the arterial pulse signal returns. Record this number as the brachial systolic pressure for the measured arm.
- Step 6: Repeat steps 3 to 5 for the contralateral arm and record that brachial systolic pressure.
- Step 7: For *ankle measurements*, locate both the dorsalis pedis (DP) and posterior tibial (PT) pulses using the Doppler probe.
- Step 8: Position the Doppler probe steady over the DP pulse and inflate the ankle blood pressure cuff to 20 mm Hg above the mark where the DP pulse signal is no longer appreciated and deflate the cuff slowly until the arterial pulse signal returns. Mark this number as the DP systolic pressure for the measured side. Some patients may have congenitally absent DP pulses.
- Step 9: Repeat step 7 with the Doppler probe over the PT pulse to measure the PT systolic pressure for the measured side and record that number.
- Step 10: Apply the blood pressure cuff to the opposite ankle, and repeat steps 6 and 7 for the opposite ankle and record both numbers.
- Step 11: Use the equations shown in [Table 284.2](#) to calculate the ABI for the patient's right and left lower extremities.
- Step 12: Interpret the results as noted in [Table 284.3](#)

|   |
|---|
| <b>TABLE 284.2 CALCULATE ABI FOR RIGHT AND LEFT LOWER EXTREMITIES</b> |
|---|

|                  |   |
|------------------|---|
| <b>Right ABI</b> | Higher of the right ankle systolic pressure (DP or PT) mm Hg<br>Higher brachial systolic pressure (right or left) mm Hg |
| <b>Left ABI</b>  | Higher of the left ankle systolic pressure (DP or PT) mm Hg<br>Higher brachial systolic pressure (right or left) mm Hg  |

**TABLE 284.3 INTERPRETATION OF MEASURED ABI VALUES IN PERIPHERAL ARTERY DISEASE**

| <b>ABI VALUE</b> | <b>INTERPRETATION</b>                        |
|------------------|--|
| >1.3             | Calcified vessels                            |
| 1.3–0.91         | Normal vessel flow                           |
| 0.9–0.4          | Mild to moderate peripheral arterial disease |
| 0.39–0.00        | Severe peripheral disease                    |

*Table 284.3* outlines the interpretation of the ABI in peripheral artery disease. In trauma or other acute processes, the ABI can also be helpful to determine patients at risk for lower extremity arterial injury. In patients with lower extremity trauma, for example, ABI values  $\leq 0.9$  have a sensitivity of 87% and specificity of 97% for lower extremity arterial injury and thus warrant further imaging with angiography or operative intervention. Values  $\geq 0.91$  suggest a lower likelihood of arterial injury and a strategy of observation, repeat ABI measurements, and/or nonemergent angiography.

## KEY POINTS

- ABI is an important noninvasive test to assess arterial disease or injury in both ED and outpatient settings.
- ABI values  $\leq 0.9$  in the acutely injured lower extremity warrants further evaluation with angiography or operative intervention.

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## **DON'T MISS THE PROXIMAL FIBULA FRACTURE IN PATIENTS WITH ANKLE FRACTURE**

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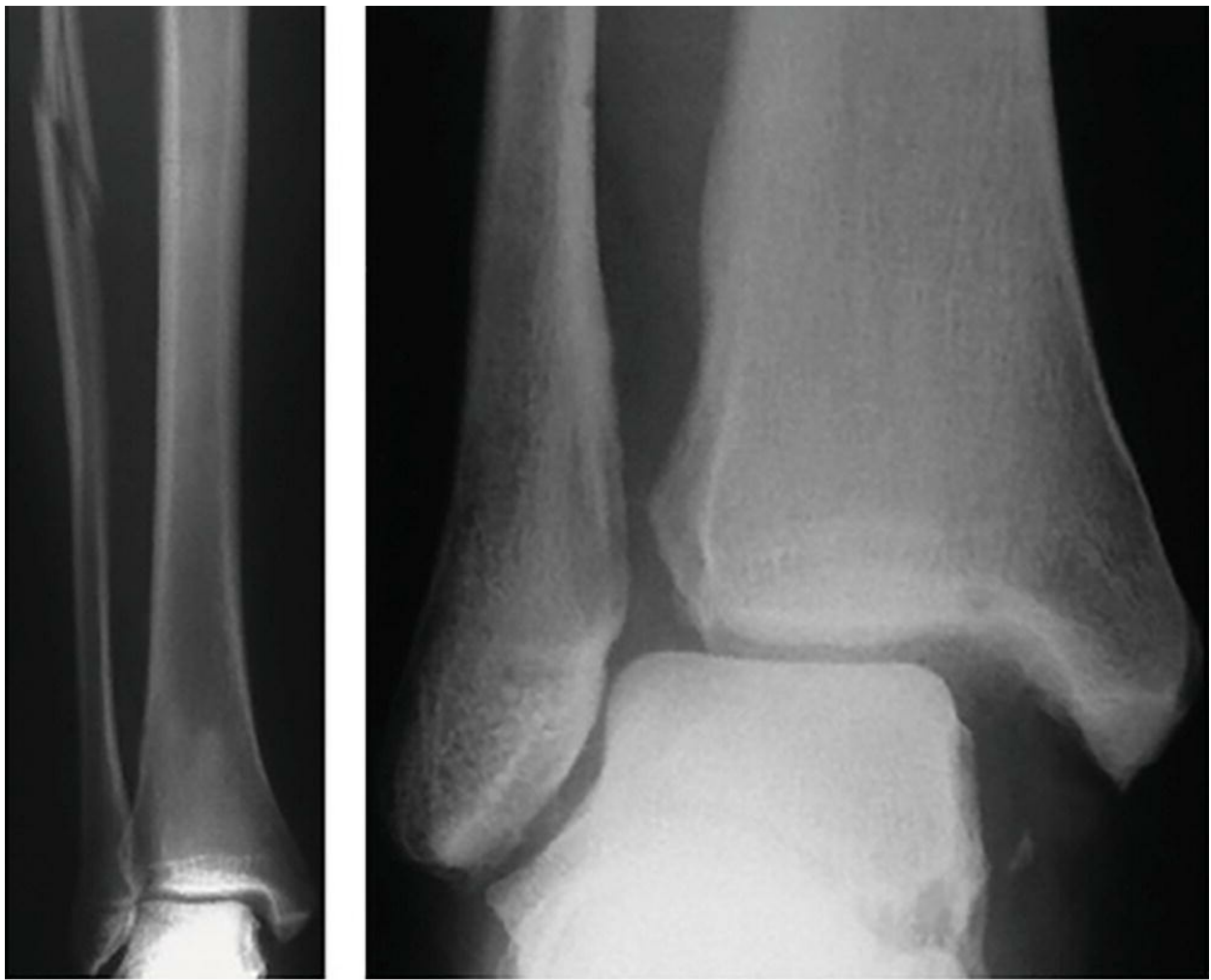
**SHAWN K. KAKU, MD, STUART SWADRON, MD,  
FRCPC**

One of the most common musculoskeletal injuries evaluated in the emergency department (ED) is the twisted ankle. Significant research has been conducted on the evaluation of the injured ankle in the ED, with the creation of rules to help aid in the differentiation between a sprain and a fracture via history and physical examination alone. However, not all ankle injuries are isolated to the distal extremity. Specifically, one rare but significant injury pattern of the ankle injury can throw the clinician off track because it also causes a fracture near the knee. It is known as the Maisonneuve fracture.

The Maisonneuve fracture is a spiral fracture of the proximal third of the fibula with disruption of the distal tibiofibular syndesmosis and associated injury. This fracture pattern is caused when an external rotation force is applied to the fixed foot. The force of the injury runs from the distal tibia, up through the interosseous membrane and ends at the proximal third of the fibula. This force creates an injury pattern that first causes an injury in the deltoid ligament and/or fracture of the medial malleolus. Next, there is a rupture of the distal tibiofibular syndesmosis with occasional fracture of the posterior malleolus. Finally, the force causes a rotational and valgus stress on the proximal fibula causing a proximal fibular fracture. The characteristic feature of the Maisonneuve fracture is a spiral or oblique fracture at the fibular neck or immediately proximal to the neck.

The mechanism of injury of the Maisonneuve fracture is most often sports related, followed by injuries caused from slipping on ice, walking or running, and finally by motor vehicle accidents and falls from height. Patients may complain of only ankle pain and resultant inability to ambulate and not complain of proximal fibular pain. This may be due to the minimal weight-bearing demands of the proximal fibula. On examination, there will be tenderness present over the deltoid ligament (on the medial side) and the syndesmosis without lateral ligamentous or distal fibular tenderness. While there will likely not be any obvious deformity or swelling over the proximal fibula, there is usually tenderness to palpation. Moreover, the patient may complain of decreased sensation to the dorsum of the foot. This is a result of an injury to the peroneal nerve as it crosses over the head of the fibula.

The lack of a historical complaint of proximal fibular pain is precisely why we need to conduct a thorough examination, extending to at least the proximal fibula (joint above and joint below!) of any patient with a complaint of an ankle injury. An x-ray of the ankle often demonstrates a widening of the distal tibiofibular joint as well as a fracture of the medial malleolus and/or posterior malleolus. However, in some cases, the proximal fibular fracture can occur with only soft tissue damage at the level of the ankle (see [Figure 285.1](#)). Only films of the tibia-fibula or knee, if ordered, will demonstrate the proximal fibula fracture. Furthermore, the talus may spontaneously reduce medially, leaving little to suspect on the basis of ankle films alone that a high fibular fracture may be present. Thus, without recognition of the injury pattern and examination of the proximal fibula, many patients with Maisonneuve fracture patterns are missed. Tibia and fibular radiographs should not necessary be obtained routinely, but should be obtained if there is bony tenderness over the proximal fibula in the setting of an ankle injury or if an ankle fracture is appreciated on x-ray.



**A**

**B**

**Figure 285.1** A Maisonneuve fracture with a proximal fibula fracture (**A**) and lateral displacement of the talus and widening of the syndesmosis (**B**). (From Court-Brown C, Heckman JD, McKee M, et al., eds. *Rockwood and Green's Fractures in Adults*. 8th ed. Philadelphia, PA: Wolters Kluwer, 2014.)

Treatment of a Maisonneuve fracture is most commonly surgical and depends on the nature of injury to the ankle mortise. Failed diagnosis can lead to long-term pain and arthritis. With proper diagnosis and management, nevertheless, the long-term functional outcome is usually good.

## KEY POINTS

- The Maisonneuve fracture is a spiral fracture of the proximal third of the fibula with associated disruption of the ankle. It presents most commonly as a sprained ankle.

- Management is most often surgical, and delays in diagnosis may result in complications.
- Diagnostic delays can occur because of a failure to examine and, if necessary, image the proximal fibula in ankle injuries.
- The absence of an actual ankle fracture in some cases (only soft tissue injury) may lead the clinician to underestimate the severity of this injury.

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## BOXER'S FRACTURE? CHECK FOR ROTATIONAL DEFORMITY!

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JENNIFER MARVIL, MD, MA

Boxer's fracture is the common name for a fifth metacarpal neck fracture, although the term is often applied to fractures of both the fourth and fifth metacarpals. Metacarpal fractures account for ~40% of hand injuries; fractures of the metacarpal neck are the most common type of metacarpal fracture with the fifth metacarpal being the most commonly injured, accounting for ~20% of all hand fractures. The highest incidence of boxer's fractures is seen in men ages ~15 to 30 years. Despite the name, boxer's fractures are not very common in trained boxers but are seen more frequently in untrained fighters.

The pattern of a boxer's fracture occurs due to a direct impact or axial load to the metacarpal head with the metacarpophalangeal (MCP) joint in flexion, often as a result of a punch thrown against a solid surface. This fracture is inherently unstable due to the loss of proximal stabilizing forces to the metacarpal head, as the collateral ligaments insert proximally on the sides of the metacarpal head and distally onto the phalanx. The loss of proximal stabilizing forces and the typical direction of impact onto the dorsum of the metacarpal head generally lead to volar angulation of the metacarpal head.

Clinically, a boxer's fracture can be diagnosed by swelling, deformity, or tenderness over the metacarpal or depression of the fifth MCP joint. The hand should additionally be examined for skin integrity, as a laceration sustained as a result of blows to an opponent's mouth (also known as a "fight bite") can lead to serious bacterial infection and functional impairment. Radiographic characteristics of a boxer's fracture include a typically oblique fracture of the metacarpal neck with volar angulation of the distal segment and metacarpal head (see [Figure 286.1](#)).



**Figure 286.1** Boxer's fracture. Oblique radiograph of the hand shows a fracture of the fifth metacarpal (*arrow*) with volar angulation of the

distal fragment. (From Siegel MJ, Coley B, eds. *Core Curriculum: Pediatric Imaging*. Philadelphia, PA: Wolters Kluwer, 2005.)

Metacarpal neck fractures can tolerate a variable degree of angulation without functional impairment. Angulation is best assessed on the lateral radiograph of the hand. The acceptable degree of angulation is different for each digit. While the exact number of allowed angulation at each digit varies between sources, ~10, 20, 30, and 40 degrees of angulation are allowed as you move from the second to fifth metacarpal. The acceptable volar angulation is in addition to the inherent 15 degrees of volar angulation between the fifth metacarpal neck and head. More volar deformity is allowed at the fifth metacarpal because the fifth carpometacarpal joint is more mobile than the other carpometacarpal joints, allowing for more rotational capacity to facilitate opposition. Similarly, the thumb can also tolerate ~40 degrees of volar angulation for a fracture of the first metacarpal neck due to the increased rotational capacity at the first carpometacarpal joint to allow for opposition.

Yet, while fractures of the fifth metacarpal neck can tolerate a great deal of angulation, no degree of rotational deformity is acceptable. Rotational deformity leads to functional impairment due to overlap of digits resulting in decreased grip strength. Rotational deformity can be assessed by both clinical and radiographic findings. With fingers flexed at both the MCP and proximal interphalangeal (PIP) joints, all digits should point to the scaphoid tuberosity. Additionally, the nail beds of the digits should be in alignment with the digits in extension. One should suspect a rotational deformity if all the digits do not align uniformly or if there is overlap of the fourth and fifth digits with a closed fist. In addition to the clinical exam findings, radiographs of the hand should be assessed for rotation in addition to angulation. While volar angulation is best assessed on lateral films, any apparent angulation observed on an anteroposterior (AP) film represents significant malrotation. It is important to note, however, that radiographs of the hand are not very sensitive for detecting malrotation. Therefore, the provider should remain diligent in assessing for rotational deformity clinically. Fractures with malrotation require orthopedic referral for operative fixation to prevent functional impairment.

## KEY POINTS

- Boxer's fracture refers to the fracture of the fifth metacarpal neck as a



result of closed fist impact to a hard surface. This is the most common metacarpal fracture of the hand.

- While metacarpal fractures can tolerate variable degrees of volar angulation (depending on the digit involved), no degree of rotational deformity is acceptable. Clinicians should diligently assess all boxer's fractures for presence of rotational deformity clinically as radiographic findings may not be sensitive enough to detect malrotation of the distal fragment.
- Rotational deformity can be assessed by looking for overlap of the fourth and fifth digits while forming a closed fist, or by evaluating the alignment of the nail beds of all digits in extension. All fractures with rotational deformity will require orthopedic referral for operative fixation in order to prevent functional impairment due to decreased grip strength.

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# THINK OF ACHILLES TENDON RUPTURE IN PATIENTS WITH SPRAINED ANKLE

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JENNIFER FARAH, M

Sprained ankles run the gamut, some with impressive physical exam findings (e.g., diffuse edema, extensive ecchymosis) and others with only mild limitations in range of motion. Regardless, the workup may include an x-ray, which if negative for a fracture is typically followed by the application of an elastic bandage and recommendations for RICE (rest, ice, compression, and elevation) therapy. However, a very concerning diagnosis may be slipping away—one that is often not considered in what appears to be a benign ankle sprain—an Achilles tendon rupture.

The Achilles tendon is the largest tendon in the body. Unfortunately, it is also one of the most poorly vascularized, and thus, it is vulnerable to injury. It connects the gastrocnemius and soleus muscles to the calcaneus. With every step, it undergoes repetitive inversion and eversion, which can lead to an incredible amount of microtrauma and inflammation over time. Running, jumping, and other sports-related movements can exacerbate an already weakened tendon, causing further damage. Tendonitis and bursitis are common conditions, but even more concerning is a full or partial rupture of the Achilles tendon.

Given the subtlety of presentation, nearly 25% of Achilles tendon injuries are initially missed. Most patients with these injuries are men, 30 to 50 years old, and seasoned athletes are just as vulnerable as newcomers. The patient may report that he made a very sudden movement, heard a “pop,” and was unable to continue with his current activity. For example, a basketball

player may describe having his feet firmly planted while he pivoted to catch a pass. Another common description is the sensation of being “kicked in the back of the ankle.”

On examination, the provider may palpate a noticeable defect or “knot” along the length of the tendon. One may also appreciate a loss of plantar flexion or an inability to perform a toe raise. However, not all cases are this obvious, and more provocative testing may be necessary. In the Thompson test, the patient is placed supine with the affected limb flexed 90 degrees at the knee. The physician then squeezes the calf and observes if there is plantar flexion of the foot. If it is absent, this is considered an abnormal test and an Achilles tendon injury should be suspected. If there is limited plantar flexion, the physician can further investigate by placing a sphygmomanometer on the calf. With the patient in the same supine position, and the foot plantar flexed, the cuff should be inflated to 100 mm Hg. The physician then passively dorsiflexes the foot at the sole and observes for a rise in the pressure reading. With an intact tendon, the pressure should rise to 140 mm Hg. This value can vary from person to person; thus, to ensure the most accuracy, this test should also be performed on the unaffected limb to note the patient’s baseline.

It is important to remember that patients with Achilles tendon injuries may have retained plantar flexion due to the action of other surrounding muscles, such as the flexor hallucis longus and tibialis posterior. Therefore, imaging may be needed to make a definitive diagnosis. Magnetic resonance (MR) and ultrasound (US) are both very helpful. Given the high cost and limited access to an emergent MR, US is favored in the emergency department. Findings include a loss in tendon continuity or variation in echogenic texture of the tendon that could represent a partial tear. However, these findings are operator dependent and are not as sensitive as MR.

Treatment of an Achilles tendon rupture involves prolonged immobilization or surgical repair plus immobilization. The decision of which course to pursue is made in conjunction with an orthopedic surgeon and takes into consideration the patient’s level of activity. If immobilization is performed in the ED, the ankle should be immobilized in plantar flexion (splint or cast in equinus) and the patient should anticipate that the immobilization will remain for possibly 8 to 12 weeks, with the first 3 to 4 weeks as non-weight bearing. Expectations should be managed; the patient should expect a slow recovery that may not lead to full return of function.

## KEY POINTS

- Achilles tendon rupture should be on the differential diagnosis of any patient who presents with ankle pain.
- Patients with Achilles tendon injury show limited or lack of foot plantar flexion upon the provider squeezing their calf (abnormal Thompson test).
- A bedside US of the tendon can be a useful adjunct in making the diagnosis for an Achilles tendon rupture.
- Once the diagnosis is made, the ankle should be immobilized in plantar flexion (equinus), and the patient should be referred to an orthopedic surgeon for follow-up.

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## REDUCE HIP DISLOCATIONS IN A TIMELY MANNER

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**ERIK A. BERG, MD**

Hip dislocation is an orthopedic emergency that requires prompt diagnosis, evaluation and treatment. This injury primarily occurs after a high-energy traumatic mechanism such as head-on motor vehicle collision (MVC), auto-versus-pedestrian accident, major fall, and contact sports injury. Consequently, hip dislocation is frequently accompanied by life-and limb-threatening injuries. Nevertheless, providers should know that the outcome of a hip dislocation is related to the time to reduction. The earlier the reduction—which should be within 6 hours of the injury—the better the results.

The hip joint is formed by the articulation of the proximal head of the femur (the “ball”) into the acetabulum (the “socket”). Reinforced by labral cartilage, a joint capsule, and strong muscular and ligamentous attachments, the hip joint is generally very stable and requires significant force to dislodge. The medial and lateral circumflex femoral arteries provide the majority of blood supply to the femoral head. These are terminal arteries with poor collaterals. When disrupted, they render the femoral head susceptible to avascular necrosis (AVN).

Hip dislocations are classified as anterior, posterior, or central, based the position of the femoral head relative to the acetabulum. Posterior dislocations account for about 80% of all hip dislocations and most commonly occur in high-speed MVC. Patients present with a shortened leg held in adduction with the hip flexed and internally rotated. Anterior hip dislocations (10% to 15%) occur with forceful hip abduction, extension, and external rotation and may present as a lengthened leg with an abducted, flexed, and externally rotated hip. Central dislocations are very rare and result from direct impact

on the lateral thigh, forcing the femoral head through a comminuted acetabulum (a fracture dislocation of the hip).

A plain anteroposterior (AP) film of the pelvis is typically the only imaging necessary to confirm the diagnosis. The AP pelvis x-ray should be examined for any interruption of Shenton line (normally, the smooth, continuous contour of the inferior border of the superior pubic ramus and the inferomedial border of the neck of femur), which should raise suspicion for hip dislocation or femoral neck fracture. Shenton line is illustrated in [Figure 288.1](#). Additional imaging should be performed for two reasons: (1) if the patient requires a CT scan for other injuries and there is additional time to quickly obtain cuts through the acetabulum and femoral metaphysis or (2) the AP view of the femoral neck is inadequate to rule out fracture. In the second case, lateral or oblique (Judet) plain films may be useful adjuncts.



**Figure 288.1** Shenton line (dashed line). (From Pope TL Jr, Harris JH Jr. *Harris & Harris' The Radiology of Emergency Medicine*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2012.)

The goal in managing hip dislocation is reduction in <6 hours. The primary contraindication to a closed reduction is a femoral neck fracture. Other fractures of the acetabulum and femur are not contraindications, though they may render the reduction more difficult. Although reduction is within the scope of practice of emergency physicians, appropriate procedural sedation and analgesia should be employed and preparations for splinting should be made prior to the reduction. Avoid multiple attempts at reduction in the emergency department, as there is an increased associated risk of damage to the articular surface and AVN with each attempt. If the fracture is irreducible, the patient will require a trip to the operating room for either a closed reduction under general anesthesia or an open reduction.

Complications from hip dislocations include traumatic arthritis, sciatic nerve dysfunction, and most importantly, AVN of the femoral head. AVN is perhaps the most disabling sequela of hip dislocations. In order to minimize the risk for the development of AVN, reduce the hip within 6 hours of injury!

## KEY POINTS

- Consider hip dislocation in high-energy traumatic mechanisms such as MVCs or falls from height.
- Any disruption of Shenton line should raise suspicion for a fracture or dislocation.
- Reduction should be performed as soon as possible and in <6 hours to avoid the complication of AVN.

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## CHECK FOR SNUFFBOX TENDERNESS AND DON'T MISS A SCAPHOID FRACTURE

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**BENJAMIN D. MUSSER, MD**

Wrist pain is a very commonly encountered chief complaint in the emergency department (ED), and emergency providers must be aware of the high rate of scaphoid fractures among patients with acute wrist pain. The scaphoid bone is the most commonly broken wrist bone, and fracture is often the result of axial loading of the wrist via a fall onto an outstretched hand.

The scaphoid is a crescent-shaped bone in the wrist and the largest of the proximal carpal bones. Its anatomy can be remembered by dividing it into thirds: the proximal pole, the middle third (waist), and the distal pole. An understanding of the unique blood supply to the scaphoid helps us to best treat these injuries. The scaphoid receives its vascular supply from the palmar carpal branch of the radial artery. This branch enters the scaphoid at the distal end and then travels retrograde toward the proximal pole. The waist and proximal pole of the scaphoid are therefore dependent on an intact *distal* blood supply, which will frequently become compromised after fracture.

When a suspicious history for scaphoid fracture exists, clinical exam of the wrist will often help solidify the diagnosis. Examination will classically reveal tenderness and/or swelling over the anatomical snuffbox (best tested with the wrist in slight volar flexion and ulnar deviation). Tenderness will also be elicited with palpation over the scaphoid tubercle on the volar aspect of the hand and with axial loading of the thumb metacarpal. All three of these quick bedside maneuvers correlate with the presence of a scaphoid fracture.

When ordering x-rays to look for a scaphoid fracture, the series will need to have a dedicated scaphoid view. Obtaining this view involves placing the patient's hand in full pronation with as much ulnar deviation of the wrist as tolerated. A clear fracture line is obviously indicative of fracture, but more subtle findings such as obliteration or displacement of the scaphoid fat pad may indicate the presence of a fracture as well. A study by Waeckerle et al. showed that plain radiographs performed after an acute injury have a false-negative rate of up to 20%. Therefore, if clinical suspicion for a scaphoid fracture is high despite negative radiographs, empiric immobilization is recommended. Repeat wrist radiographs can be obtained in 1 week in such cases. This will allow more time for any fracture to present itself, while keeping the patient immobilized in the interim. If faster diagnosis is necessary, as in the case of athletes who would otherwise return to play, Carpenter et al. showed that magnetic resonance (MR) and computed tomography (CT) have excellent sensitivity and specificity for identifying scaphoid fracture even in the acute setting.

Splinting or casting after a scaphoid fracture focuses on immobilization of the scaphoid and usually requires placement of a thumb spica cast or splint. Duration of immobilization varies, but longer immobilization is required for more proximal fractures because of the higher risk of avascular necrosis and nonunion. This increased risk can be explained by the pattern of blood supply described above. The compromised blood supply to the proximal pole in a proximal or neck fracture can impede healing and greatly increase the likelihood of avascular necrosis.

If there is significant concern for complication after scaphoid fracture, orthopedic referral from the ED is warranted. Several criteria for urgent orthopedic referral include

- Fractures of the proximal pole
- Greater than 1 mm displacement of fracture segments
- Delayed presentation. Langhoff et al. showed that patients with delayed presentation of 4 weeks after their initial scaphoid injury had as high as 40% rate of nonunion.

In patients with uncomplicated scaphoid fractures without evidence of nonunion, there is no evidence showing a long-term benefit of surgical intervention compared to immobilization. However, there is a slight decrease in time to return to work with surgical intervention as shown in a study by Bond et al. (8 weeks vs. 15 weeks in a small study involving 25 patients).



## KEY POINTS

- Scaphoid fracture is the most common fracture involving the carpal bones of the wrist. The most common mechanism of injury is a fall onto an outstretched hand.
- Snuffbox tenderness, tenderness upon palpation of scaphoid tubercle, and pain with axial loading of the thumb metacarpal are physical exam findings that should increase suspicion for scaphoid fracture.
- It is important to immobilize the wrist and thumb with a thumb spica splint if clinical suspicion for scaphoid fracture exists, even despite negative radiographs.

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## **CALCANEAL FRACTURE? DON'T MISS A SPINAL INJURY!**

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**SARA KHAGHANI, MD, MPH**

Calcaneus fractures are a source of debilitating disease and are important to quickly identify. The incidence of calcaneal fracture is 11.5 per 100,000, with a male to female predominance of 2.4:1. These fractures have high rates of acute and long-term complications. They are often the result of high-impact trauma, most often occurring after an axial load injury on the foot after a fall (or jump!) from 6 feet or more. In one study, 72% of calcaneus fractures occurred after a fall.

Plain films are the initial study of choice for confirming the presence of a calcaneal fracture. Lateral and axial (Harris) views of calcaneus as well as an anteroposterior (AP) view of the foot are indicated. Additional views or computed tomography (CT) may be required to further define the extent of the fracture. CT is often used in cases where the fracture is intra-articular. Intra-articular calcaneus fractures have a poorer prognosis because they extend into the weight-bearing subtalar joint. An orthopedic surgeon should be consulted for these injuries for proper management; urgent referral is usually sufficient.

Open fractures, any neurovascular injury, fractures with dislocation and acute compartment syndrome are potential complications that require emergent orthopedic surgical consultation. It is important to be aware of skin necrosis, which can commonly occur when there is posterior displacement of the calcaneus.

Initial management of calcaneus fracture includes elevation of the limb about the level of the heart, as well as icing of the injury. A bulky compression dressing (also referred to as a bulky Jones splint) should be

applied. Of course, analgesia is indicated. Frequent skin exams are required to assess for skin necrosis and compartment syndrome. Surgical repair may be indicated for displaced and comminuted fracture patterns involving the articular spaces of the foot.

It has been shown that up to 50% of patients with calcaneal fracture have other associated injuries. The most commonly seen concomitant injuries are to the lower limbs (13.2%) and thoracolumbar spine (6.3%). In addition, about 5% of patients have bilateral calcaneal fractures. It is therefore important to do a careful head-to-toe examination of the axial and appendicular skeleton and a neurologic exam that includes motor function, sensation, reflexes, and position sense. In patients with a high-force mechanism or signs of injury on examination, imaging of the axial skeleton (e.g., x-ray or CT of the thoracolumbar spine) is appropriate. CT has a higher sensitivity for detecting spinal fractures.

## KEY POINTS

- Calcaneus fracture results from high-impact trauma such as a fall from significant height, and is associated with other injuries.
- Carefully examine the other calcaneus, thoracolumbar spine, and lower limbs.
- Adequate initial radiographic views to assess for calcaneal fracture include lateral and axial (Harris) views of the calcaneus, as well as an AP view of the foot.
- Clinicians should be aware of possible compartment syndrome and skin necrosis of the foot as a result of calcaneus fracture. Emergent orthopedic consultation is indicated if either appears imminent.

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# **BEWARE OF BENIGN-APPEARING HIGH-PRESSURE INJECTION INJURIES**

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**JENNIFER FARAH, MD**

“Looks can be deceiving” has never been truer than when identifying high-pressure injection injuries. Presentation may be limited to a small puncture wound or a vague neurologic deficit. Thus, we must be diligent in investigating the details of the injury. Common causes of high-pressure injection injury are paint guns, grease guns, and other occupational instruments. Men are more likely to present with such injuries, with an average age in the 30s. Often, the patient was exploring a clogged nozzle when the injury occurred. This is why the index finger of the nondominant hand is the most common digit injured. Even the most experienced skilled workers are not immune to these mishaps.

Delay in presentation is common, as the injected material may not initially cause discomfort. It may take many hours before the patient feels compelled to visit the emergency department. As the agent disturbs the surrounding tissues, inflammatory processes take effect, and this leads to further swelling and pain. While nearby blood vessels and nerves become compressed, the finger will appear more edematous, tense, and pale, and often the patient will report pain and paresthesia. Limitations in range of motion and compromised perfusion may be appreciated on examination, with only a small punctate lesion noted grossly. The foreign material will then use neurovascular bundles and tendon sheaths as a highway to travel more proximally. One case report found that an auto mechanic suffered pneumomediastinum as a consequence of compressed air injected into his hand. This serves as a reminder, yet again, that although these patients may

have wounds that are unimpressive at first glance, the level of underlying damage can be devastating.

The material injected should, of course, be determined. Systemic reviews have noted that organic solvents, such as paint, paint thinner, fuels, and oil tend to be more caustic and therefore more likely to lead to amputation. On the other hand, the inflammatory response to air or water is less robust.

Injections should be further explored initially with x-ray. The clinician may note a radiopaque substance, for example with paint, revealing the extent of injury. Also, there may be an increased lucency indicating that water or air has disrupted the tissue. Administration of broad-spectrum antibiotics and tetanus prophylaxis is advised. The role for steroids is still in question; their effect on dampening the inflammatory process may be beneficial but they have not been shown to reduce the incidence of amputation. Ultimately, no medical therapy should delay the ultimate intervention, which is surgical debridement.

Time to surgical debridement is of the utmost importance, as amputation rates can be as high as 50%. Studies have shown that the risk of amputation is less if the patient undergoes debridement within 6 hours of injury. Surgical exploration is needed to not only eradicate the offending chemical and irrigate the necrotic tissue but also decompress surrounding nerves and vessels. There are minimal published data regarding the overall functional outcome of these patients, but any efforts to reduce the chance of amputation should be welcomed; patients who often suffer from these injuries usually are dependent on maintaining proper dexterity more than most.

## KEY POINTS

- High-pressure injection injuries often have a benign appearance on examination, yet this mechanism of injury indicates significant underlying tissue injury.
- Plain radiographs of the affected extremity may help reveal the true extent of the underlying injury.
- Though broad-spectrum antibiotics and tetanus prophylaxis are advised in these cases, time to surgical exploration and debridement is the most important factor in preventing a devastating outcome and minimizing the chance of amputation.

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# LISFRANC INJURY: DANGER IN THE MIDFOOT

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**LEE PLANTMASON, MD, MPH**

Lisfranc injury is an injury to the tarsometatarsal (TMT) joint complex. It constitutes roughly 0.2% of all fractures, and is considered one of the causes of significant disability from injuries to mid-and forefoot. It can occur from both low-energy mechanisms, such as falls from standing and athletic injuries, and high-energy mechanisms, such as falls from height, crush injuries of the foot, and motor vehicle collisions. While history and physical examination with careful attention to plain radiographs are important, plain radiographs are not always diagnostic due to overlapping bones, especially on the lateral view. Up to 20% of these injuries may not be accurately diagnosed on initial plain radiographs, making them a high-risk injury for emergency physicians to miss.

A brief review of foot anatomy helps in understanding Lisfranc injuries. The forefoot is composed of five metatarsal bones and their associated phalanges. The midfoot also consists of five bones: three cuneiforms (medial, middle, and lateral), the cuboid, and the navicular. The Lisfranc or TMT joint consists of the articulations between the metatarsals and the three cuneiforms and cuboid, all very critical to the stability of the foot. The Lisfranc joint is composed of three longitudinal components, the medial column (medial cuneiform and first metatarsal), middle column (middle and lateral cuneiforms with the second and third metatarsals) and the lateral column (the cuboid and the fourth and fifth metatarsals). The Lisfranc ligament runs from the plantar medial cuneiform to the base of the second metatarsal, while the second through fifth metatarsals are interconnected through a series of intermetatarsal ligaments, thus connecting the medial column to the lateral four metatarsals and serving as the primary soft tissue support of the TMT

articulation.

Lisfranc injury results from both direct and indirect trauma; most commonly direct trauma includes crush injuries associated with significant soft tissue injury, vascular insufficiency, and compartment syndrome of the foot. Most common indirect injury patterns are due to forced external rotation, axial loading of a foot in plantar flexion, or twisting of an axially loaded foot in fixed equinus position where forced abduction of the forefoot causes dislocation of second metatarsal and lateral metatarsal displacement. For example, when first described by French war surgeon Jacques Lisfranc de St. Martin, a soldier who fell off his horse with his foot still in the stirrups might have sustained this type of injury.

In general, patients with Lisfranc injuries tend to present with midfoot tenderness, edema, and an inability to bear weight. Forefoot and midfoot edema with plantar ecchymosis are considered pathognomonic for Lisfranc injury. Other physical exam findings suggestive of TMT joint injury include the “piano key test,” where one can induce pain or subluxation by dorsiflexion and plantar flexion (or abduction and adduction) of the first and second metatarsals.

Anteroposterior (AP), oblique, and lateral x-ray views are utilized for initial assessment of Lisfranc injury. The AP is used to assess the alignment of the first and second TMT joints by determining if the medial border of the second metatarsal lines up with that of the middle cuneiform. The oblique is used to assess the other TMT joints, by determining if the medial border of the fourth metatarsal lines up with the cuboid. Lateral radiograph may show dorsal dislocation or subluxation between the first and second metatarsals. Additionally, avulsion fractures of the second metatarsal or medial cuneiform, also known as the “fleck sign,” or >2 mm of diastasis between the first and second metatarsals suggest TMT joint injury (see [Figure 292.1](#)).





**Figure 292.1** AP x-ray of patient with a Lisfranc injury. The medial border of the second metatarsal does not line up with that of the middle. Note the “fleck sign,” representative of an avulsion of the Lisfranc ligament from the base of the second metatarsal. (From

Altchek DW. *Foot and Ankle Sports Medicine*. Philadelphia, PA: Wolters Kluwer, 2012.)

If a Lisfranc injury is clinically suspected despite normal imaging, either weight-bearing or stress views of the foot should be obtained including oblique, lateral, and AP views. Any displacement  $> 2$  mm between the first and second metatarsals is diagnostic of ligamentous Lisfranc injury. Comparison x-ray views of the unaffected foot can also aid in the diagnosis. Given the pain and discomfort of obtaining these views, the patient should receive analgesia prior to obtaining films and receive clarification on why additional images are needed.

Management of Lisfranc injuries depends on the degree of displacement. Minimally displaced ( $<1$  mm between the first and second metatarsals) Lisfranc injury is generally managed conservatively with a non-weight-bearing splint, rest, ice, and elevation with outpatient orthopedic evaluation at 2 weeks. In general, these patients will remain in a controlled ankle movement (CAM) boot for 6 to 10 months before starting physical therapy. On the other hand, displaced Lisfranc injuries ( $>2$  mm of displacement) are unstable and require orthopedic consultation from the ED with specific attention placed on monitoring for compartment syndrome. These injuries will usually require transarticular fixation or arthrodesis depending on the degree of injury and will not bear weight for 8 to 15 weeks.

## KEY POINTS

- Lisfranc injury occurs from injury to the TMT joint, and if not diagnosed and properly managed can lead to significant disability to the patient. Midfoot tenderness and edema, plantar ecchymosis, and inability to bear weight on examination should raise suspicion for Lisfranc injury.
- When a Lisfranc injury is suspected, foot radiographs can reveal malalignment of the TMT joints and/or the fleck sign. Weight-bearing or stress views, or comparison views of the unaffected foot can aid in diagnosis when initial radiographs are nondiagnostic and clinical suspicion for the injury remains high.
- Patients with Lisfranc injury should be splinted with instructions to remain non-weight bearing. Minimally displaced injuries can be urgently referred to orthopedics as outpatient, while displaced injuries with significant soft tissue edema should be monitored for

compartment syndrome and be evaluated by an orthopedic surgeon in the ED.

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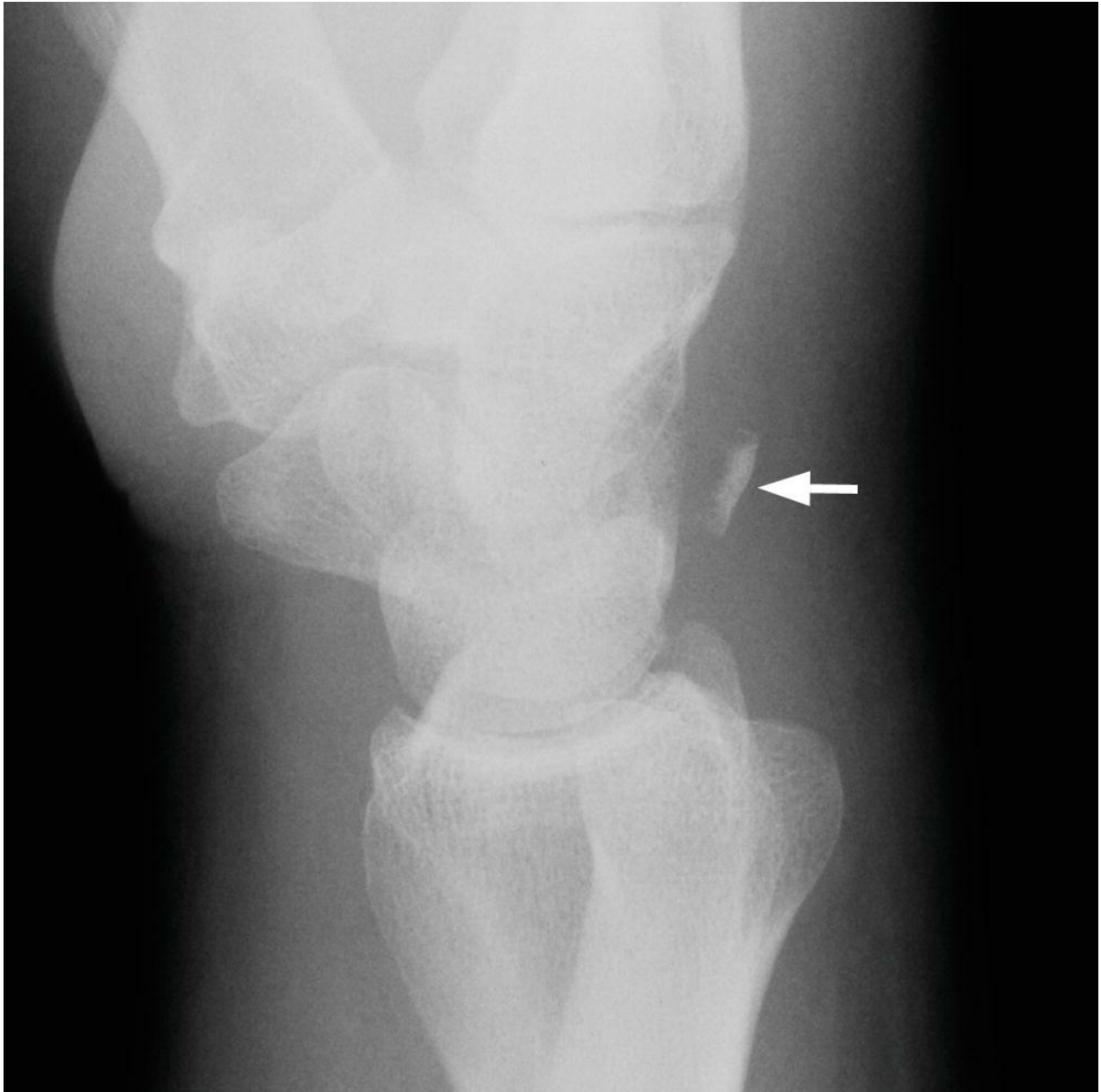
## THE DORSAL CHIP: IS IT A TRIQUETRAL FRACTURE?

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CAROLINE BRANDON, MD

Carpal bone fractures are very common, with scaphoid fracture constituting the majority of injuries. The second most common type of carpal fracture is the triquetral fracture, constituting from 15% to 19% of all carpal fractures. Triquetral fractures occur by mechanisms similar to scaphoid fractures. Yet they can be very subtle in presentation and on x-ray, so it is important to be on the lookout for these injuries.

There are three major types of triquetrum fracture: dorsal cortical, body, and volar. Dorsal cortical fractures occur with a fall onto a dorsiflexed and ulnarly deviated hand. Patients present with midwrist tenderness, edema, and pain with range of motion. Anteroposterior (AP) and oblique wrist x-rays generally show no acute abnormality. Yet, the lateral x-ray of the wrist generally shows a dorsal fleck of bone. (see [Figure 293.1](#)) As a result, this pattern of fracture is also commonly referred to as a *dorsal chip* fracture. This fracture rarely requires surgery and is usually splinted with a volar splint and then subsequently casted. Return to full function is expected in 6 to 8 weeks.



**Figure 293.1** Triquetral fracture. Lateral radiograph shows displaced avulsion fragment (*arrow*) with overlying soft tissue swelling. (From Chew FS. *Skeletal Radiology*. 3rd ed. Philadelphia, PA: Wolters Kluwer, 2010.)

Triquetral body fractures constitute 3% of triquetral fractures, and they can be further divided into 3 subcategories. Sagittal body fractures are associated with crush injuries and axial dislocations. Medial tuberosity fractures occur from a direct blow to the triquetrum. Transverse body fractures are associated with a high-speed trauma, perilunate injuries, or other comminuted fractures. As these injuries are the result of high-energy mechanisms, the physical examination of such wrists typically reveals significant swelling and limitation of range of motion. Plain radiographs usually demonstrate these various fracture patterns, as well as ligamentous

instability. Nonetheless, if the x-rays are negative and the clinical suspicion (based on mechanism of injury and degree of soft tissue injury) remains high, computed tomography (CT) of the wrist is appropriate. These injuries often require surgical repair with open reduction; orthopedic surgery should be consulted from the emergency department.

Volar fractures have been described as avulsion injuries involving the palmar ulnar triquetral or lunotriquetral ligaments. These fractures are unstable because of the ligamentous involvement and require expeditious orthopedic referral. They are difficult to diagnose with plain radiographs and often require magnetic resonance (MR) imaging to evaluate the extent of the instability. When significant doubt exists as to the presence of a serious injury and plain films are nondiagnostic, it is appropriate to immobilize the wrist with a volar splint and arrange close orthopedic follow-up. If left untreated, these patients develop arthritis and ligamentous instability.

Triquetral fractures (and other carpal bone fractures) are associated with fractures of the distal radius. Therefore, one important pitfall for the emergency physician is to forget to look for additional fractures in the same extremity once one is identified.

Overall, triquetral fractures can be tricky to diagnose. Risks associated with missed or delayed diagnosis include nonunion and vascular compromise. If the mechanism of injury and physical examination are concerning for a triquetral fracture but you don't see a fracture on x-ray, consider advanced imaging. If a distal radius fracture is present, make sure to examine the hand to rule out concurrent carpal bone fractures.

## KEY POINTS

- The triquetrum is the second most common of the carpal bones to be fractured.
- Dorsal chip fractures can be diagnosed on lateral radiographs of the wrist as a fleck of bone at the dorsum of the wrist. These do well with simple immobilization.
- Triquetral body and volar fractures can result from high-energy mechanisms and are often associated with other bony or ligamentous injuries. These are serious injuries that can result in morbidity. Prompt orthopedic evaluation is important.
- X-rays can be nondiagnostic for triquetral fractures. If injury is suspected, patients can have their wrist immobilized in a volar splint until orthopedic evaluation and/or advanced imaging can be obtained.

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## **LUNATE AND PERILUNATE DISLOCATIONS: PICK THESE UP ON INITIAL PRESENTATION!**

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**TODD SCHNEBERK, MD, MA**

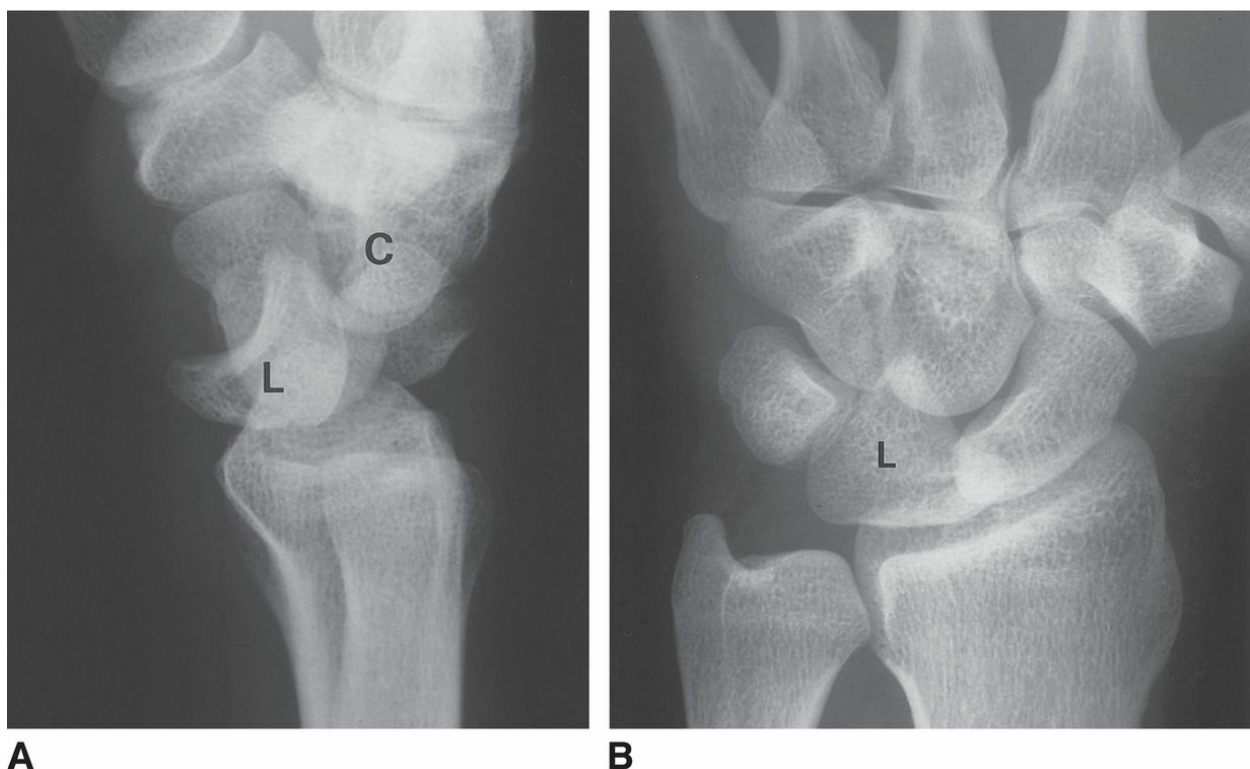
Perilunate and lunate dislocations, although differing in radiographic appearance, represent similar injuries along a continuum of carpal instability with lunate dislocation representing a more severe ligamentous disruption than does perilunate dislocation. Both dislocations are rare and can be easily missed. They are also similar in terms of management and treatment considerations. These two injuries embody the high-stakes nature of emergency medicine; not recognizing them and failing to facilitate an emergent reduction and expedient referral to a hand surgeon will result in poor patient outcomes.

Perilunate and lunate dislocations occur most commonly in young men around the age of 30, typically, as a result of hyperextension of the wrist with ulnar deviation from a high-energy mechanism (motor vehicle accident, fall from height, etc.). They should be considered as part of the differential diagnosis of wrist pain in any patient with a fall onto an outstretched hand (FOOSH) mechanism of injury. On physical examination, the wrist will demonstrate significantly decreased range of motion and profound swelling with diffuse tenderness to palpation. Patients are at risk of developing compartment syndrome due to significant tissue edema. Associated median nerve deficits can also be present, increasing diagnostic suspicion for dislocation as the lunate lies dorsal to the median nerve and the displacement that occurs in both injuries can result in subsequent nerve compression. Moreover, multiple injuries may be present. For example, perilunate dislocations are often accompanied by scaphoid fractures; initial focus on a



seemingly isolated scaphoid fracture can result in the clinician missing a more serious dislocation.

The lateral x-ray is the most useful view to diagnose a wrist dislocation and to distinguish lunate from perilunate dislocation. Though it can be somewhat confusing, the identification of these injuries is based on the appearance of the lunate. In the lateral x-ray of a normal wrist, the radius, lunate, and capitate are collinear with capitate on top of lunate, which in turn sits on top of radius. Perilunate dislocation can be thought of as a capitate translocation dorsally off of the top of the lunate. The lunate still articulates with the radius inferiorly with only a slight volar rotation. On the other hand, lunate dislocation represents a more extensive carpal and ligamentous disruption. In this dislocation, the lunate is profoundly volar rotated and displaced, no longer articulating with the radius. Yet, the capitate can remain in nearly anatomic position collinear with the radius. This appearance is known as the classic “spilled teacup” sign. The posteroanterior film, although usually less useful than the lateral, will often show the lunate appearing much more triangular than normal. This is named as the “piece of pie” sign, and while it is beneficial if identified, it can occur in both lunate and perilunate dislocations. Therefore, it is of little help when it comes to distinguishing between the two compared to the lateral view ([Figures 294.1](#) and [294.2](#)).



**Figure 294.1** Lunate dislocation. **A:** The lateral radiograph of the wrist shows the lunate (L) tipped off of the distal radius, whereas the

capitate (C) seems to be normally aligned in relation to the radius yet is dislocated from the lunate. **B:**Anteroposterior view shows a pie-shaped lunate (L) rather than a lunate with a more rhomboid shape. A pie-shaped lunate on an anteroposterior view is diagnostic of a perilunate or lunate dislocation. (From Brant WE, Helms CA, eds. *Brant and Helms Solution*. Philadelphia, PA: Wolters Kluwer, 2005.)



**Figure 294.2** Perilunate dislocation. Although the lunate (L) is in a normal relationship to the distal radius, the capitate (C) and the remainder of the wrist are dorsally displaced in relation to the lunate. (From Brant WE, Helms CA, eds. *Brant and Helms Solution*. Philadelphia, PA: Wolters Kluwer, 2005.)

Once recognized, closed reduction is indicated prior to sending the patient home. The technique for closed reduction of both of these dislocations is

similar, and it is accomplished using axial traction and hyperextension at the wrist with volar pressure to the lunate, followed by flexion with continued pressure on the lunate. The use of finger traps can facilitate reduction in both cases. Specialist consultation is appropriate in cases of where there are multiple injuries and when the emergency staff is unable to perform the reduction. Once reduction is achieved, the wrist should be splinted in a sugar tong splint and referral made to a hand surgeon urgently as almost all cases will require surgical fixation. Even after initial surgery these injuries can go on to develop median neuropathy, posttraumatic arthritis, or chronic ligamentous instability, often requiring further surgical intervention and further underscoring the need for proper management on the initial presentation.

## KEY POINTS

- It is important to consider lunate and perilunate dislocations in patients with significant swelling, reduced wrist range of motion after high-mechanism, and FOOSH injuries.
- *Perilunate dislocation* appears as disruption of the lunocapitate articulation, usually with dorsally displaced capitate. *Lunate dislocation* appears as volar displacement and angulation of the lunate off of the radius, also known as the “spilled teacup” sign.
- Following recognition of these injuries, timely reduction and referral for surgical fixation are necessary, as functional disability can be devastating if these injuries are missed or subsequent treatment is delayed.

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# RED FLAGS FOR INTIMATE PARTNER VIOLENCE AND HUMAN TRAFFICKING

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**KRISTI STANLEY, MD**

Intimate partner violence (IPV) is a serious and potentially life-threatening health problem. It is defined as any physical, psychological, or sexual aggression or violence by a current or former partner or spouse. Over 30% of women and 21% of men will encounter IPV in their lifetime. Emergency physicians (EPs) must be aware of IPV and its warning signs, as it is challenging to identify and manage. While patients may present to the emergency department (ED) for treatment of injuries resulting from IPV, they are often reluctant to self-report the abuse, due to embarrassment or fear. Thus, IPV is both underrecognized and underreported.

The ED encounter is an opportunity for IPV screening to identify patients who are victims of violence and to prevent further injury. EPs are likely to come across victims of IPV in their daily practice, as these patients seek care for acute injuries. Because victims can be difficult to identify, universal screening is more likely to identify victims of IPV than targeted screening. In performing screening, it is important that we use open-ended questions and nonjudgmental tone and body language. The EP should be prepared with a response for the patients who screen positive for IPV.

When interviewing the patient, take the history with the patient alone to allow for disclosure of IPV if present. A partner or spouse who appears defensive, or who answers for the patient or will not allow the patient to be alone with health care providers is a red flag for abuse. The EP should assure the patient of confidentiality, excluding mandatory reporting of violence as

required by each state. Signs that alert the provider to consider IPV include history inconsistent with pattern of injury, bruises in various stages of healing, evidence of delay to seeking care, and minimization of injuries. In addition, victims of IPV are at higher risk for psychiatric illness, substance abuse, and suicide. Screening should take place after intoxication has resolved. All patients who screen positive for IPV should be asked about suicidal and homicidal ideation. More subtle presentations of IPV include patients who have vague symptoms, chronic pain, or multiple visits to the ED.

On physical examination, injuries may not be readily obvious, so it is important to examine the entire patient. Victims of abuse have injuries that are more centrally located, that is, face, neck, breasts, abdomen, and genitalia, than the peripheral injuries seen in accidental trauma. EPs should also recognize defensive injuries, such as injuries to the ulna or hands as victims fend off blows or to the bottoms of the feet from kicking the assailant.

Women are at particular risk of harm from IPV during pregnancy. During pregnancy, IPV may escalate. Women abused during their pregnancy are at higher risk of continued abuse postpregnancy. A delay to seeking prenatal care may be an indicator of IPV. Abdominal and genital trauma in pregnancy should prompt providers to inquire about IPV.

In addition to IPV, EPs may encounter victims of human trafficking. These patients experience social isolation and are often unfamiliar with their specific whereabouts. Language barriers may be present, with the trafficker often serving as a translator. Red flags include patients who do not speak for themselves and who do not control their own identification documents. When these patients do provide a history, it may be coached, rehearsed, and inconsistent. They may appear anxious and fearful and avoid eye contact. The EP may identify malnutrition and untreated health conditions on examination, in addition to signs of physical and/or sexual abuse.

When IPV or human trafficking is suspected, ensuring the patient's safety is vital. The EP should involve social workers or community advocates to provide resources to the patient and to develop a plan for long-term safety. Prior to discharge, the patient and the health care team must decide if the patient is safe to go back to his or her home. The patient should be reassured that the abuse they have sustained is not deserved, nor is their abuser's behavior part of a healthy relationship. It is a physician's responsibility to report IPV, as required by their state. All 50 states mandate reporting of child abuse and elder abuse; however, mandated reporting of IPV varies among states. Physicians must also report child abuse if it is

disclosed during screening for IPV.

## KEY POINTS

- Intimate partner violence (IPV) and human trafficking are common, life threatening, and underrecognized.
- A history inconsistent with pattern of injury, bruises in various stages of healing, evidence of delay to seeking care, and minimization of injuries are all red flags for IPV.
- Be especially careful with patients who do not speak for themselves or who do not control their own identification documents—they may be victims of human trafficking.
- Physicians are mandated reporters of child abuse, elder abuse, and, depending on the jurisdiction, IPV.

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## SECTION XX

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# PROCEDURES/SKILLS/AL



# **SEDATION PEARLS AND PITFALLS: PROCEDURAL SEDATION IN THE EMERGENCY DEPARTMENT**

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**CHIDUBEM ILOABACHIE, MD AND DENA REITER, MD**

Procedural sedation, generally defined as a treatment strategy for the administration of sedative or analgesic medications to intentionally suppress a patient's level of consciousness, is common in today's emergency departments. Though it is remarkably safe when performed by well-trained emergency physicians, unanticipated adverse events can certainly occur. Consequently, careful attention in the preparatory phase as well as pharmacologic selection is critical.

## **PATIENT ASSESSMENT**

Preparing a patient for procedural sedation starts with a thorough history and examination. This is to exclude elements that may make procedural sedation more dangerous, and allow the clinician to make appropriate pharmacologic choices. In particular, the interview should focus on potential allergies or intolerances to drugs that are being considered for use. The patient's comorbidities must be considered as well. For example, obstructive lung disease, sleep apnea, pregnancy status, anatomical anomalies, and cardiac disease should be considered in order to determine the safest plan of action. These patient characteristics must be considered when anticipating potential adverse events regarding ventilation and perfusion. Particular regard to body habitus, facial hair, dentition, and Mallampati score should be given. Assigning an ASA (American Society of Anesthesiologists) class gives a general idea of how well the patient may tolerate sedation. It is critical that

the patient's consent is obtained when possible both for the procedure and the sedation.

## **PHARMACOLOGIC SELECTION**

Understanding of pharmacology is critical in order to perform the sedation safely and smoothly. One must consider both the level of sedation desired (ranging from anxiolysis to deep sedation) as well as the desired duration of the sedative effects. A patient undergoing cardioversion only requires seconds of sedation, whereas a patient undergoing a complex fracture reduction may need over 30 minutes of adequate sedation and analgesia.

Specific patient characteristics should also drive medication selection. A hypotensive patient is not an appropriate candidate for propofol, and a patient prone to seizures may not be an ideal candidate for etomidate, which may lower the seizure threshold. Patients who are agitated or hypertensive may not be good candidates for ketamine. Recent evidence suggests that combinations of agents—each used at lower dosages than when given independently—can mitigate the adverse effects of each and synergistically improve analgesic and sedative effects. This has been shown particularly true for ketamine and propofol.

## **ANTICIPATE ADVERSE EVENTS**

As mentioned above, the physical examination should also be used to determine the relative ease with which the patient's airway and respiratory functions can be supported if the level of sedation is deeper than anticipated.

The patient should have reliable intravenous access with two separate sites being preferred over one. This is to administer reversal agents or crystalloid boluses in the event of cardiopulmonary compromise. It is also reasonable that the patient receive supplementary oxygen to mitigate against hypoxia should apnea occur. Emergency medicine literature suggests that capnometry is useful in early identifying cases of apnea where hypoxia has not yet occurred. While it remains equivocal if identification of such episodes is clinically superior to physician observation of the patient, there is no disadvantage associated with its use.

To start, the patient or health care proxy consent should have been made in advance, whenever possible with a clear statement of the risks and benefits of the proposed treatment plan as well as alternative courses of action. The environment should be prepared with all supplies needed for airway management. This should include, but is not limited to a bag valve mask,

suction, endotracheal tubes, and a laryngoscope. The specific state of “readiness” of these devices will typically depend on the performing physician’s risk tolerance. Generally speaking, however, the adage that something is not available “unless it’s within arm’s reach and in its operative configuration” may be applied. All monitoring including capnography, telemetry, oxygen saturation, and blood pressure readings should be easily visible to all concerned parties.

If any of the above measures reveal concerns, it should be reconsidered whether procedural sedation is the best approach for the patient compared to analgesia alone, regional anesthesia, or admission or execution in an operating room or procedure suite with the assistance of an anesthesiologist.

## **CONSIDER THE AVAILABLE RESOURCES**

When preparing staff, it is advisable to have a medical provider dedicated specifically to sedating and monitoring the patient and another dedicated specifically to the procedure that required the sedation in the first place. That is to say, procedural sedations should always have at least two health care professionals including a nurse to monitor the patient while the physician executes the procedure. Notably, the monitoring should be protocolized (e.g., blood pressures every 3 minutes) and paired with documentation of the proceedings.

As with any advanced treatments in medicine, a “timeout” should precede procedural sedation. Though there is not a standard approach to a timeout, consider including in the process introduction of parties, confirmation of the patient’s identity, and confirmation of the procedure to be executed. With respect to procedural sedation, the timeout should also include multiparty confirmation of the dosage of medication to be administered. This recommendation reflects the relative unfamiliarity of sedative, dissociative, and anesthetic drugs (e.g., compared with analgesics, antibiotics, etc.) and the potentially deadly consequences of a misplaced decimal point when reading a solution concentration or calculating a dosage per body weight. Closed-loop communication when administering these powerful medications is more important than ever.

Once the procedure is complete, it is important for observation to continue until the patient has demonstrated a clear trajectory of recovery. Depending on local policies, it is reasonable for the physician to leave the bedside once that recovery trajectory has passed the point where cardiopulmonary support would be necessary. Notably, this does not necessarily coincide with return to baseline but may be at levels consistent with minimal sedation (anxiolysis) or even moderate sedation (conscious

sedation). Discharge from the emergency department, however, should not occur until return to baseline. If patients are to be discharged, there should be a responsible family member or another concerned adult accompanying the patient home.

## KEY POINTS

- Unique patient characteristics such as comorbidities, allergies, anatomy, and type of procedure being performed must all be considered when selecting the appropriate sedation strategy.
- Patients undergoing sedation must be closely monitored using telemetry, blood pressure readings, pulse oximetry, and possibly continuous capnometry. Clinical observation is also paramount.
- Plan for adverse events, and have rescue equipment readily available.
- Available resources as well as predicted risk of sedation must be considered. Certain sedations are more safely performed in a controlled operating room under the care of an anesthesiologist.

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# **CAPNOGRAPHY IN THE ED: QUALITATIVE OR QUANTITATIVE MONITORING? FOR CPR AND A WHOLE LOT MORE**

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**ARUN NAIR, MD, MPH**

Capnography has become a mainstay in most emergency departments. It is a useful tool in confirming endotracheal tube (ETT) placement, assessing a patient's disease severity, and their response to our interventions. But what does capnography and end tidal CO<sub>2</sub> (EtCO<sub>2</sub>) actually mean?

Capnography devices measure the partial pressure of CO<sub>2</sub> at the end of the tidal breath—which is when the alveoli finally empty. Therefore, capnography is a combined measure of ventilation, circulation, and metabolism as it reflects both the CO<sub>2</sub> being delivered to and exiting from the lungs. Furthermore, there are two types—quantitative and qualitative. The most common qualitative device is a colorimetric device: that little purple square that is placed at the end of the ETT that turns to yellow if there is CO<sub>2</sub> in the expired air. Quantitative measurements usually involve a nasal cannula hooked up to a monitor that gives both a value and waveform of the CO<sub>2</sub> in the expired air. So, what are some of the pitfalls of capnography?

## **CONFIRMING PLACEMENT**

Purple or yellow, seems pretty simple, right? Actually, these devices aren't binary. They are pieces of litmus paper that have three different color/CO<sub>2</sub>

ranges: purple <3 mm Hg, tan 3 to 15 mm Hg, yellow >15 mm Hg. Like most litmus paper, these devices are reflecting a change in acidity which is a proxy for CO<sub>2</sub>. Therefore, any acid that inadvertently comes into contact with the paper can produce a false positive. Gastric contents are notorious culprits when vomitus enters the ETT. But also be aware that the epinephrine and lidocaine we use in code situations is acidic as well and can also give a false positive. Animal models have shown that even large volumes of ingested carbonated beverages can give a false positive with esophageal intubation. If there is any question of contamination, either switch to a new block or change to a quantitative device.

How about false negatives? In a patient not in cardiac arrest, successful ETT placement should yield a bright yellow within the first few breaths. But, if no CO<sub>2</sub> is being delivered to the lungs (massive PE, no circulation) then there will be no CO<sub>2</sub> in the exhaled air despite a successful intubation, so no color change. Sometimes the paper turns that frustrating purple tan that makes you doubt yourself even though you're sure you saw the cords.

## FOR PROCEDURES

Your hospital's policy may now require you to use wave capnography for procedural sedation which can be very frustrating when it means waiting for extra personnel and equipment when you could have just done the darn thing already, right? Why the additional monitoring? Remember, capnography measures the patient's ventilation and this combined with pulse oximetry is a robust measure of the patient respiratory status. If the patient becomes over sedated, the decreasing respiratory drive causes hypoventilation which in turn yields a build-up of CO<sub>2</sub> that you can see in real time. A sudden flatline can mean severe laryngospasm if there is still chest rise or complete apnea if there's not. The respiratory rate measurement on most monitors can be poor indicators of actual ventilation as they are affected by patient movement and sweat, and have no reflection of tidal volumes. Studies have shown that patients on passive oxygenation with SO<sub>2</sub> > 97% that appear to be spontaneously breathing can have EtCO<sub>2</sub>s in the 70s to 90s! This build-up sometimes can be seen minutes before the hypoxia ever registers on pulse oximetry. The use of quantitative capnography can guide you to decrease sedation, provide extra stimulation, or even give a few BVM breaths to restore respiratory drive. Early recognition and intervention can hopefully preclude the need for securing the airway when the patient's sats suddenly drop into the 80s while everyone was fixating on that prosthetic hip ortho was reducing.

# EVALUATING RESPIRATORY DISTRESS

Too often we are content with just knowing the EtCO<sub>2</sub> number and completely disregard the waveform, but there's a lot of info here! So, just what does that yellow hump show? The initial upstroke of the wave represents the alveoli rapidly emptying their CO<sub>2</sub> into the main airway. The almost vertical line should then take a near 90-degree angle into the plateau as CO<sub>2</sub> is effectively delivered to the nose throughout the exhale. When this angle is instead obtuse and rounded like a shark fin, it means that the alveoli are not effectively emptying—likely secondary to an obstructive process (COPD/asthma/bronchospasm). This waveform can then be helpful in differentiating COPD and CHF exacerbations as well as a patient's response to neb treatments without having to wait for the blood gas to result. If the waveform isn't squaring up and the EtCO<sub>2</sub> is increasing, you should probably be considering NIPPV or intubation as alveoli are continuing to collapse and the patient is tiring out. If such monitoring is used on an obtunded critically ill patient, a normal waveform and CO<sub>2</sub> of 35 to 45 mm Hg indicates a patent airway, spontaneous breathing, and adequate perfusion of the lungs in the matter of seconds!

## FOR CPR

If your current CPR routine does not include the use of EtCO<sub>2</sub>, you're really doing yourself a disservice.

- EtCO<sub>2</sub> gauges the effectiveness of CPR: rising EtCO<sub>2</sub> indicates rising pulmonary and coronary perfusion and higher EtCO<sub>2</sub> values positively correlate with adequate chest compression depth, ROSC and survival.
- EtCO<sub>2</sub> is the earliest indicator of ROSC: the restarted heart causes a rapid increase in EtCO<sub>2</sub> as the built-up CO<sub>2</sub> in now reperfused tissues is being effectively delivered to the lungs. This bump can be seen well before a pulse is felt.
- EtCO<sub>2</sub> can show when to stop: EtCO<sub>2</sub> < 10 mm Hg after 20 minutes of good CPR accurately predicts death.

Bottom line: Not using EtCO<sub>2</sub> is the common error.

## KEY POINTS

- End tidal CO<sub>2</sub> devices measure the partial pressure of CO<sub>2</sub> at the end of the tidal breath.
- Qualitative capnography uses colorimetric changes but can be subject to inaccuracies if there is any acidic contamination.
- Quantitative capnography can alert you to impending inadequate ventilation.
- Quantitative capnography waveforms can be used to gauge response to therapy in patients with obstructive respiratory processes.
- Quantitative capnography can be used in CPR to help detect reperfusion.

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# TO TRANSFUSE OR NOT TO TRANSFUSE

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**DEBRA RAVERT, MD**

In the past century, blood transfusion has gone from experimental to readily available therapy for conditions ranging from acute blood loss to chronic anemia. For several decades, there was very little consensus about when transfusion was indicated, and therefore there was much variability in practice. The TRICC (Transfusion Requirements in Critical Care) trial in 1999 changed the face of transfusion by providing evidence that transfusing at a hemoglobin threshold of 7 g/dL was as good as, if not better than, using a threshold of 10 g/dL in critical care patients. This study triggered a wave of additional investigations aimed at validating numerical thresholds for various specific indications. Almost all subsequent studies have validated the safety of using a threshold of 7 g/dL, with the notable exception of patients with acute coronary syndrome. However, most of the trials since 1999 have explicitly excluded active bleeding, which is a common presentation in the emergency department. So, what are the indications for transfusion in the emergency setting? We will review three broad categories: acute and ongoing bleeding, symptomatic anemia, and hemoglobin thresholds.

Active bleeding is an attention grabber, and arguably more common in the emergency department than anywhere else in medicine. Laboratory values have less utility when the patient has or is suspected to have very recent or ongoing bleeding. Patients with active bleeding lose whole blood (plasma and red cells), so it takes several hours for their hemoglobin to equilibrate; a patient who comes in immediately after a 2-L blood loss from a stab wound may have an initial hemoglobin of 14 but it may drop to 8 after a few hours. In this setting, absolute hemoglobin has less value and clinical judgment, largely based on history and physical, becomes more important.

Key historical questions focus on possible sources of bleeding including epistaxis, hematemesis, hematochezia, melena, hematuria, menorrhagia, and injury, and on symptoms of anemia, including light-headedness, dizziness, chest pain, and shortness of breath. A thorough physical exam is especially important when there is a concern for ongoing bleeding and should encompass all potential sources of bleeding, both external and internal (including a good lung and abdominal exam). If there is concern for ongoing bleeding on history and physical, transfusion may be indicated even if tested hemoglobin is within normal limits.

Anemia can contribute to a variety of signs and symptoms. Patients may become symptomatic after a relatively rapid drop in hemoglobin (whether from blood loss or hemolysis) or after a more gradual decline. It is important to assess each patient for signs and symptoms that could be attributable to anemia. Symptoms can be very vague, including fatigue, dizziness, light-headedness and, in severe cases, syncope, but can also include angina, dyspnea, palpitations, pica, and restless leg syndrome. On physical exam, look carefully for signs such as low blood pressure, pallor, koilonychia (concave fingernails), lethargy, tachycardia, systolic flow murmur, or splenomegaly. Even when the laboratory value of hemoglobin is above 7 g/dL, if the patient's clinical picture is consistent with symptomatic anemia, transfusion may be indicated.

Numerical thresholds for transfusion also have a role in the emergency setting. Although most transfusion studies since 1999 have been done in surgical or critical care settings, their results may be extrapolated to the emergency department in many cases. In the absence of acute coronary syndrome, an absolute transfusion threshold of 7 g/dL appears to be safe, and patients with a hemoglobin below this level likely warrant transfusion even in the absence of active bleeding or overt signs and symptoms of anemia. Similarly, patients with suspected acute coronary syndrome likely warrant transfusion at a higher threshold, typically 10 g/dL. Exceptions to these numerical thresholds include patients with known pathology who are at their known baseline hemoglobin, such as patients with sickle cell disease or other hematologic or oncologic disease. These patients may be at a higher risk than the general population for sequelae of frequent transfusion (such as iron overload) and should therefore be transfused more judiciously and, if possible, in consultation with their primary provider.

In recent years, there has been more emphasis in the medical community on using blood products sparingly. In conjunction with the Choosing Wisely campaign, the American Board of Internal Medicine has recommended avoiding transfusion in hemodynamically stable patients without active bleeding whose hemoglobin is >7 g/dL. Blood transfusion is not a benign

intervention and is associated with real morbidity and mortality as well as health system cost. Understanding when transfusion is indicated and when it can be safely deferred is critical. Most of the studies informing our understanding of transfusion thresholds were conducted in surgical or critical care settings, but the lessons learned in those environments can inform our decisions in the emergency department. In cases of active bleeding, clinical judgment and patient symptom profile must prevail. If patients are symptomatic, absolute numbers are less important, and it is appropriate to consider transfusion. In stable patients without symptoms of acute coronary syndrome, a hemoglobin threshold of 7 g/dL is likely appropriate for most cases.

## KEY POINTS

- Blood transfusion is not a benign intervention.
- Laboratory data alone is not sufficient, especially in cases of acute and ongoing bleeding.
- Assess patients carefully for signs and symptoms of ongoing bleeding and symptomatic anemia.
- Only consider transfusion in asymptomatic patients with hemoglobin below validated thresholds: 7 g/dL for most, threshold for active acute coronary syndrome (ACS) less clear.

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# **TRANSFUSION CONFUSION: TYPES AND MANAGEMENT OF TRANSFUSION REACTIONS**

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Blood product transfusions play an important role in the management of patients in the emergency department with indications ranging from traumatic hemorrhage to reversing anticoagulation. Blood products include packed red blood cells, platelets, cryoprecipitate, and fresh frozen plasma. The first method to avoid a transfusion reaction is to ensure that the situation warrants a transfusion. After the emergency physician makes the decision to transfuse a blood product, the patient needs to be monitored for signs of a transfusion reaction. These reactions can range from a benign febrile reaction to life-threatening hemolysis. If a transfusion reaction is suspected, then the transfusion must be stopped and the blood bank notified.

Febrile nonhemolytic transfusion reactions (FNHTRs) present with an increase in temperature of at least 1°C from baseline accompanied by chills, rigors, and discomfort. These symptoms usually occur within 2 hours of initiating the transfusion. These are caused by a release of cytokines either from the transfused blood or from native cytokines released as a reaction to transfused leukocyte antigens. The risk of FNHTRs can be decreased, but not eliminated, by using leukoreduced red blood cells. Many physicians premedicate with acetaminophen and diphenhydramine to prevent FNHTRs, but recent studies have suggested that premedication does not decrease the rate of FNHTRs and may mask symptoms of more severe transfusion reactions.

Allergic transfusion reactions (ATRs) present with urticarial rashes and

pruritus within the first 2 hours of a transfusion. These reactions are caused by IgE-mediated activation of mast cells and basophils resulting in histamine release. While these reactions tend to be mild, patients can experience anaphylactic reactions with bronchospasm, respiratory distress, angioedema, and hypotension. These severe reactions occur most commonly in IgA deficient patients. The management ranges from antihistamines to epinephrine and vasopressors depending on the degree of allergic symptoms.

Hemolytic transfusion reactions (HTRs) present with chills, fever, and flank pain progressing to hypotension, renal failure, and disseminated intravascular coagulation. HTRs are caused by immune-mediated destruction of donor red cells by preexisting recipient antibodies with ABO mismatch serving as the classic example. The severity of the reaction depends on the amount of transfused blood with as little as 200 mL of transfused blood being fatal. The treatment for HTRs involves aggressive hydration and supportive care.

Transfusion-associated circulatory overload (TACO) commonly presents in patients with impaired cardiac function with the transfusion resulting in hydrostatic cardiogenic pulmonary edema and respiratory distress. Common presenting symptoms include dyspnea, orthopnea, tachycardia, and a wide pulse pressure. Laboratory and imaging investigations typically reveal an elevated *N*-terminal Pro-BNP and a chest radiograph with bilateral infiltrates. The treatment of TACO involves volume reduction with diuretics, supplemental oxygen, and in severe cases noninvasive positive pressure ventilation.

Transfusion-related acute lung injury (TRALI) and TACO have similar initial presentations and can be difficult to distinguish. These symptoms include dyspnea and respiratory distress. TRALI is caused by increased vascular permeability from donor antileukocyte antibodies leading to noncardiogenic pulmonary edema. Imaging commonly reveals bilateral infiltrates on chest radiograph. The management of TRALI is supportive and may require intubation and mechanical ventilation for hypoxemia as well as the initiation of vasopressors for sustained hypotension.

Transfusion-associated sepsis (TAS) occurs when a blood product contaminated with bacteria is transfused with symptoms of fevers and hypotension that may progress to septic shock. Most commonly, TAS is associated with platelet transfusions since they are stored at room temperatures providing an ideal environment for bacterial replication. Treatment includes broad-spectrum antibiotics and hemodynamic support with fluids and vasopressors as needed. Additionally, blood cultures should be obtained from both the patient and transfused blood sample.

Transfusion-associated graft versus host disease occurs when an immunocompromised host is transfused with immunocompetent lymphocytes. The transfused lymphocytes then mount an immunologic response against the host's cells. Symptoms begin about a week after transfusion including rash, fever, elevated liver enzymes, jaundice, and gastrointestinal symptoms. There is no treatment, and this reaction is universally fatal. In order to prevent this reaction, transfusions for immunocompromised hosts are irradiated to eliminate donor lymphocytes.

Overall, there are a wide variety of transfusion reactions that can occur ranging from benign to life threatening. The most important step is early recognition, and then stopping the transfusion.

## KEY POINTS

- The best way to avoid a transfusion reaction is to avoid unwarranted transfusions.
- If a transfusion reaction is suspected, then the transfusion must be stopped and the blood bank notified.
- Premedicating before transfusions may mask symptoms of severe transfusion reactions.
- TACO and TRALI are often difficult to distinguish, but both treated with supportive care.
- TAS is most commonly seen with platelet transfusion.

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## ARTHROCENTESIS TIPS

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**CHRISTINE MLYNAREK, MD AND ASHLEY SULLIVAN, MD**

Arthrocentesis, the aspiration of synovial fluid from a joint space, is a valuable diagnostic and therapeutic tool in the emergency department. The most common indication for arthrocentesis is to aid in the diagnosis of an acutely swollen atraumatic joint, which carries a broad differential, the most serious of which is septic arthritis. Septic arthritis is a rapidly destructive microbial invasion of the joint space, which can lead to significant morbidity and mortality. Up to 75% of patients develop substantial functional disability, and fatality rates remain at 5% to 15%. Thus, it is critical to avoid delays in diagnosis. The emergency medicine physician must maintain a high index of suspicion and a low threshold to perform an arthrocentesis.

The highly vascular synovial fluid lacks a protective basement membrane and allows bacteria to easily migrate into a joint space through hematogenous spread, direct inoculation, or contiguous spread. *Staphylococcus aureus* and streptococcus account for 91% of cases. Once in the joint space, the body's inflammatory response causes rapid destruction of cartilage and surrounding structures. Certain patient populations are at higher risk including the elderly, immunosuppressed, as well as those with diabetes, alcoholism, IV drug use, prosthetic joints, and chronic arthritis. Furthermore, acute flares of chronic inflammatory joint conditions, including the crystal arthropathies, can present similar to septic arthritis and may even present concomitantly with septic arthritis. More importantly, these individuals are predisposed to joint infection and require a lower threshold for performing arthrocentesis.

The classic presentation of septic arthritis is an acutely swollen, hot joint with an associated fever and decreased joint range of motion. Although it is typically thought to involve a single joint, most commonly the knee or hip, up to 22% of cases have a polyarticular presentation. Though septic arthritis



can present with a systemic inflammatory response, it is important to note that fever, elevated peripheral WBC, and ESR are nonspecific, and their absence does not rule out the diagnosis. Studies have shown that an elevated ESR carries the highest sensitivity (96%), followed by fever (57%), and peripheral leukocytosis (48%).

Arthrocentesis allows for synovial fluid analysis including joint white blood cell count (jWBC), Gram stain, and culture. Though most sources designate a “positive” result if jWBC is greater than 50,000 cells/mL, a significant proportion of patients can have a jWBC below that value. Furthermore, joint aspirate culture only identifies the causative organism in ~65% of patients. Thus, the gold standard of diagnosis remains the clinical suspicion of an experienced physician, and conversely, a negative Gram stain and culture should not exclude the possibility of septic arthritis. Patients who are believed to have septic arthritis must be promptly treated with antibiotics and arrangements made for surgical washout.

Arthrocentesis is a relatively benign procedure with no absolute contraindications and few relative contraindications, including overlying cellulitis, bacteremia, and severe coagulopathies. Use of anticoagulants is not a contraindication to joint aspiration. Potential complications of arthrocentesis include iatrogenic infection and injury to neurovascular structures. Ultrasound reduces these risks by allowing direct visualization of the joint effusion and additionally decreases the time required and the number of attempts to perform.

Arthrocentesis can be accomplished using one of three approaches: the landmark approach, the indirect ultrasound-guided approach, and the direct ultrasound-guided approach. The landmark approach relies on a thorough understanding of joint anatomy, and may be difficult in those with obese body habitus or distortion of classic anatomical landmarks. This approach may also miss other important diagnoses, such as abscesses and hematomas. The indirect ultrasound-guided approach relies on locating the target effusion using ultrasound visualization, approximating the depth to the target, and marking the skin for needle entry. Once the target has been acquired and depth approximated, the ultrasound is no longer used for procedural guidance. Conversely, the direct ultrasound-guided approach relies on dynamic and continuous visualization of needle entry into the target space. It is this latter approach that is preferred, as it results in greater accuracy and a decreased complication rate.

When performing the direct approach, the patient should be positioned in the supine position, except during shoulder arthrocentesis when the patient should remain upright. A high-frequency linear transducer should be used for

the elbow, wrist, knee, ankle, and foot. A low-frequency transducer, such as the curvilinear probe, should be used for the shoulder and hip. A 20-gauge spinal needle (3.5 inches) is recommended for both the shoulder and knee joints, while a 22-gauge spinal needle (3.5 inches or 1.5 inches) can be used for smaller joints. The clinician should hold the needle in the dominant hand and maintain control of the probe with the nondominant hand. Once the area of interest is centered on the screen, the needle is inserted into the skin at equidistance away from the probe and tracked along its trajectory to the joint space. The needle should ideally be visualized with an in-plane approach and the entire length of the needle identifiable throughout the procedure.

Septic arthritis is a medical emergency, and physicians should maintain a low threshold for arthrocentesis in patients presenting with a hot swollen joint. Direct, ultrasound-guided arthrocentesis is superior to the traditional landmark technique.

## KEY POINTS

- Emergency medicine physicians must rule out septic arthritis in patients presenting with an atraumatic, acutely swollen joint.
- Chronic inflammatory joint diseases increase a patient's risk of developing septic arthritis.
- Ancillary lab tests should be considered within the context of the clinical scenario and should not be used alone to rule out septic arthritis.
- Ultrasound-guided arthrocentesis is preferred over the traditional landmark-based approach, with the direct ultrasound approach with in-plane needle visualization to be utilized whenever possible.

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# **LUMBAR PUNCTURE AND THE CHAMPAGNE TAP**

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**CHELSEA WILLIAMSON, MPAS, PA-C**

Performing a lumbar puncture is not the most difficult procedure in the emergency department, but it can quickly escalate into a frustrating and time-consuming experience. This chapter discusses high-yield tips and current evidence to help attain that “champagne tap.”

## **ASSESS THE NEED FOR AND YOUR ABILITY TO PERFORM A LUMBAR PUNCTURE**

Determine if a lumbar puncture is the correct course of action and obtain consent. There are reasons not to proceed with the lumbar puncture including, but not limited to, the following reasons: the patient or family surrogate does not give consent, contraindications are present, or the time to set up equipment and obtain required imaging will lead to delayed treatment and increased patient anxiety. Relative contraindications include the following: increased intracranial pressure (ICP), usage of anticoagulation medication, infection overlying needle insertion site, or the presence of obstruction caused by previous spinal surgery.

## **DO NOT DELAY ADMINISTERING ANTIBIOTICS IN ORDER TO PERFORM A LUMBAR PUNCTURE**

Once a lumbar puncture is indicated, time is of the essence in many cases. If meningitis is on the differential diagnosis, empiric antibiotics should be given soon, ideally within 30 minutes and should not be delayed for

cerebrospinal fluid (CSF) sampling. Antibiotic administration prior to CSF sampling has been shown to reduce culture yield, and this issue is magnified as time lapses between antibiotic administration and CSF sampling. However, cell counts and gram stain should not be affected, and blood cultures may also aid the clinician in isolating the pathogen.

## **DETERMINE IF A HEAD CT IS NECESSARY**

Intracranial imaging is usually obtained via computed tomography (CT) to rule out increased ICP. If a lumbar puncture is scheduled to be performed in this scenario, the risk of causing brainstem herniation is increased. Determining the risk of ICP may help to obviate the need for head CT, and allow the clinician to obtain CSF in a more timely manner. As such, criteria exist to rule out the presence of increased ICP without the need for a head CT. If all criteria are negative, there is a 97% negative predictive value of the patient experiencing increased ICP. These criteria include age > 60 years, immunocompromised status, history of CNS disease, seizure within 1 week of presentation, presence of papilledema, or current neurologic symptoms consistent with altered mental status or cerebral ischemia.

## **ENSURE YOU ARE FULLY EQUIPPED AND PREPARED PRIOR TO STERILIZATION**

Aim to maximize efficiency and reduce patient anxiety by avoiding the need to break sterility once the procedure is under way. Make sure that all equipment is readily available at the bedside. Confirm that you have placed all orders and printed any necessary labels. Open the LP tray to make sure it contains all the necessary pieces. It is also good to have on hand additional supplies that are not included in the kit but are commonly used, such as extra spinal needles, extra lidocaine, an extra sample tube if clinically indicated, and saline flush syringes.

## **A LITTLE FENTANYL GOES A LONG WAY**

Once patients understand that they will receive a dreaded “spinal tap,” their anxiety levels can be very high. In a procedure where patient compliance and remaining still is necessary, anxiety can be a limiting factor in obtaining fluid samples. Unless otherwise contradicted, a small dose of fentanyl will act as an anxiolytic and anesthetic. The effects of fentanyl are short acting and will fade soon after the procedure has been completed. The clinician may choose

to use a short-acting benzodiazepine such as midazolam in conjunction with an opioid, or as an alternative form of anxiolysis.

## **POSITION, POSITION, POSITION**

Whether the patient is in a lateral recumbent position or seated upright, the most important factor is that the hips and shoulders are aligned in the same plane and the back is arched. Getting the patient to maintain this position throughout the procedure is paramount for success. It is helpful for an assistant to aid the patient in maintaining the correct positioning. Landmarks may be easier to identify if the patient is seated upright. Have the patient lean over a table/tray for comfort. However, be aware that CSF pressure can only be measured when the patient is in a lateral recumbent position.

## **KNOW WHEN TO USE ULTRASOUND GUIDANCE TO FIND LANDMARKS**

It has been shown that failed lumbar punctures correlate most with higher body mass index (BMI). This is due to excess surrounding tissue, which increases difficulty in palpating appropriate landmarks to determine the proper needle insertion site. Evidence demonstrates that if a patient's BMI is 30 kg/m<sup>2</sup> or above, using ultrasound will improve a provider's ability to find appropriate landmarks by twofold. While this will aid in determining landmarks in an overweight or obese patient, remember that over palpation alone, ultrasound does not improve identifying pertinent landmarks in a patient with a BMI under 30 kg/m<sup>2</sup>.

## **NO PROVEN INTERVENTION CAN PREVENT POST-LUMBAR PUNCTURE HEADACHE**

As many as one-third of all patients who receive lumbar punctures will complain of a postprocedural headache beginning 1 to 2 days after the procedure is performed. Many prevention methods have been studied including lying supine over a pillow immediately after the procedure, preprocedural IV fluid bolus, and/or early ambulation, but none of these have proven to be of significant benefit.

### **KEY POINTS**

- Determine if an LP is the correct course of action, obtain consent, and ensure you are prepared prior to sterilization.
- Do not delay antibiotics in order to perform LP.
- Unless otherwise contradicted, a small dose of fentanyl will act as an anxiolytic and anesthetic.
- If a patient's BMI is 30 kg/m<sup>2</sup> or above, using ultrasound will improve your ability to find appropriate landmarks.
- No proven intervention can prevent the post-lumbar puncture headache.

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# **TAPPING THE BELLY: PARACENTESIS IN THE EMERGENCY DEPARTMENT**

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**THAER AHMAD, MD AND LEONARD BUNTING, MD**

It is not uncommon for patients with ascites to present to the emergency department and require paracentesis. The etiology of ascites can be diverse, but the majority of patients presenting to the ED will have underlying liver pathology. According to one study in the American Journal of Gastroenterology, delaying paracentesis can increase a patient's risk of mortality by more than twofold, while conversely performing a paracentesis within 12 hours of presentation can increase short-term survival.

As fluid in the abdominal cavity increases, patients can present with certain complaints and salient features on physical exam. These include shortness of breath, abdominal pain, nausea and vomiting, dullness to percussion, shifting dullness, and a positive fluid wave test. However, small volumes of ascites may not be evident on physical exam.

The indications for emergency department paracentesis are important for clinicians to understand. A paracentesis can be therapeutic, diagnostic, or both. Indications include suspicion for spontaneous bacterial peritonitis (fever, abdominal pain or tenderness, worsening encephalopathy, diarrhea, etc.), first-time ascites, respiratory compromise, and tense ascites concerning for an abdominal compartment syndrome.

The absolute contraindications to a paracentesis are disseminated intravascular coagulation and an acute abdomen requiring surgical evaluation. Other considerations include issues that may increase the risk of collateral damage (distended urinary bladder or bowel, pregnancy, abdominal



adhesions) and signs of abdominal wall cellulitis. Patients with platelet count  $< 20,000/\mu\text{L}$  and  $\text{INR} > 2.0$  should have these issues addressed prior to the procedure.

Proper technique for a paracentesis begins with an understanding of the landmarks. The most common puncture sites are below the umbilicus and lateral to the rectus muscles. Areas of dullness to percussion may help confirm the presence of fluid. Consider rolling the patient towards the puncture to increase fluid volume at the site.

The two sensate structures in the needle path are the skin and peritoneum. Once the optimum site has been located, a 25-gauge or smaller needle is used to anesthetize the superficial skin and deep peritoneum. The aspiration needle is then inserted at a 45-degree angle and advanced while aspirating until ascitic fluid is obtained. Peritoneal fluid should be sent for analysis in first-time ascites and if there is concern for infection.

In the emergency department removing small volumes of fluid from the abdominal cavity ( $<100\text{ mL}$ ) can be done safely. As the aspirated volume increases, attention to hemodynamic stability becomes critical. Fluid should be removed slowly to minimize the possibility of hypotension, tachycardia, electrolyte imbalances, and renal impairment. Albumin administration can also help reduce the frequency of reactions. The dose of albumin is 8 g per liter of ascitic fluid removed.

Paracentesis is a safe procedure when performed correctly with a reported complication rate of  $<1\%$ . The complications of paracentesis can be divided into hemorrhagic and nonhemorrhagic categories.

Hemorrhagic complications are rare but pose the most serious risk. Patients with these complications can present in overt shock or have more subtle complaints, such as abdominal discomfort or pain. Unrecognized or delayed bleeding can lead to hemodynamic instability and become life-threatening. Hemorrhagic complications include abdominal wall hematomas, inferior epigastric artery injuries, and hemoperitoneum. Patients who develop a significant rectus sheath hematoma should be investigated for an inferior epigastric artery injury and pseudoaneurysm. Both of these will usually occur below the arcuate line. These patients should be referred for a contrast-enhanced CT and possible embolization. Hemoperitoneum may be an acute or delayed complication. It occurs due to the drop in intraperitoneal pressure from the paracentesis, which can lead to mesenteric variceal rupture. A diagnostic paracentesis that demonstrates bloody fluid may assist in the diagnosis, and further imaging in the form of CT, ultrasound, or angiography may direct therapy.

Nonhemorrhagic complications are generally more benign, but not

negligible. Paracentesis poses a risk of small bowel perforation and iatrogenic bacterial seeding, both of which can lead to secondary infection and abscess formation. Patients showing new signs of peritonitis, fever, rigors, or unexplained symptoms of shock following paracentesis should be evaluated with imaging, surgical consultation, and/or repeat paracentesis.

Perhaps the most frustrating complication for an emergency physician is the persistent leakage of peritoneal fluid from the puncture site. To minimize this complication, consider using a small needle gauge or the Z-track technique. The Z-track technique entails putting downward traction on the skin at the puncture site prior to advancing the needle. Once fluid is aspirated, the skin is released to form a seal around the needle tract.

Mechanical complications of paracentesis can be reduced and first puncture success improved with the use of ultrasound guidance. Ultrasound can help detect the location of the largest pocket of fluid and identify structures to avoid such as the liver, spleen, bowel, and bladder. Scanning the paracolic gutters (caudal to the inferior pole of the kidney) provides a comprehensive visualization of the fluid. Noting the distance to the center of the pocket of fluid to drain allows for accurate determination of how far the needle can safely be advanced.

## KEY POINTS

- A paracentesis is typically performed below the umbilicus in the left lower quadrant where dullness to percussion is greatest.
- The patient is placed in a recumbent position and rolled towards the desired puncture site.
- The Z-track technique and smaller needle gauge help reduce the risk of persistent peritoneal fluid leaks.
- The epigastric arteries are usually found along the lateral third of the rectus; avoiding these areas will help reduce potentially lethal complication of paracentesis.
- Use of ultrasound will help identify what structures to avoid and where to place the needle for highest first pass success.

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## CAREFUL WITH THAT TAP: ACCESSING THE VP SHUNT

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**DERRICK ASHONG, MD**

Intracranial shunts have long been used for the management of elevated intracranial pressure. By the beginning of the mid 19th century, the many iterations (ventriculoperitoneal, -venous, -pleural, and -ureteral shunts) of these cerebrospinal fluid (CSF) diverting instruments had been invented. For the emergency medicine practitioner of today, intracranial shunts are associated with a myriad of disease complaints that require diagnosis and treatment. For the pediatric or adult patient, delay in diagnosis can lead to serious morbidity. Understanding the indications for placement of a VP shunt as well as the signs and symptoms associated with malfunction are vitally important for the management of a patient with this device. Additionally, learning how to access a shunt is essential for the optimal management of a decompensating patient with a likely VP shunt obstruction.

The primary reason for placement of a VP shunt is due to management of elevated intracranial pressures. In most instances, this is caused by hydrocephalus typically diagnosed during childhood. Most modern devices have four components—a proximal and a distal catheter, a reservoir, and a valve. The proximal catheter is typically placed into the right ventral horn of the lateral ventricle of the brain. This catheter is tunneled under the skin where it is connected to a valve (typically found behind the patient's ear or on the occiput). The valve is in place to ensure unidirectional flow from the ventricles, through the valve, and finally to the distal catheter. The valve contains a reservoir where CSF can be sampled or removed. While some valves have a fixed rate of CSF flow, many are programmable based on patient need. The distal catheter is again tunneled subcutaneously down the neck and chest where its end is emptied into the peritoneum.

Following placement of a VP shunt, the two major complications include infection or shunt malfunction. Infection typically occurs within the first 6 months of placement with typical bacteria being those associated with skin flora (*Staphylococcus epidermidis*, *Staphylococcus aureus*) and gram-negative bacilli from the peritoneal cavity. Shunt malfunction is typically due to an obstruction, with further delineation based on where the defect developed. Proximal obstruction can be caused by tissue debris (esp. the choroid plexus) or fibrosis. Distal (catheter) obstructions typically are due to thrombus, infection (i.e., peritonitis), or catheter migration or fracture.

Indications for tapping the shunt will primarily be driven by a high clinical suspicion for disease based on history and physical exam. Practitioners should assess for a history of fever, nausea, vomiting, and change in diet, activity level, or bowel /urinary habits. For pediatric patients, caregivers are an invaluable and important resource to assess a change from baseline as symptoms may be more subtle. The physical exam should focus on evidence of fever, papilledema, ptosis, change in gait, abdominal pain, fontanelle fullness, or “setting sun” sign, where patients may have a persistent downward gaze. The shunt should also be inspected, looking for CSF leak around the valve site, coiling of the catheter, or cellulitis over the valve site.

Imaging studies such as head CT or MRI (the latter more common in pediatric patients) should be completed to determine whether symptoms are a by-product of over- or underdrainage. Should an MRI be done, it is vitally important to involve the patient’s neurosurgeon, as the MRI magnet may disable the shunt’s programmed flow rate in some models. An x-ray shunt series can also be obtained to determine any faults along the proximal or distal catheters, although there is some controversy regarding its utility.

If clinical suspicion remains high, CSF analysis is critical, and with it, the need to tap the shunt. To do so, ensure the patient has no overlying skin infection or bleeding diathesis. Then, gather sterile gel, hair clippers, sterile gloves, povidone-iodine solution, a 25-gauge needle(s), a three-way stopcock, a CSF manometer, CSF specimen tubes, and wound dressing. First, one must palpate a few centimeters distal to the shunt entry point in the skull to find the firm shunt reservoir (usually behind the ear or along the occiput). Hair above the site can be moved away with gel or clipped. Using sterile technique, the area surrounding the reservoir should be cleaned with a povidine-iodine solution and allowed to fully dry. Anesthetics such as topical cream or 1% lidocaine can be applied to the skin following draping. Next, a 25-gauge butterfly needle should be slowly advanced into the reservoir until there is CSF return. The CSF manometer and three-way stopcock should also be connected to determine opening pressure. For accurate readings, the

manometer should be at the level of the patient's ear. When finished, the needle should be removed slowly and gentle pressure applied to the site, followed by appropriate wound care. CSF should be collected in standard sterile tubing and assessed for xanthochromia, cell count, protein and glucose levels, as well as Gram stain and culture. Additional lab tests should be ordered based on the clinical indication.

In proximal obstruction or CSF overdrainage, CSF flow may be poor or nonexistent, necessitating an emergent intervention by neurosurgery. In cases with elevated manometer pressures, CSF should be removed until the manometer pressure is below 15 mm Hg. Of note, distal obstructions are especially likely when measured pressures are >25 mm Hg.

Complications following VP shunt tap include infection, CSF leak at the puncture site, damage to the underlying valve, and ventricular collapse. Infection can be avoided with proper aseptic technique. CSF leak decreases in likelihood if appropriate pressure is applied following the procedure for around 1 to 2 minutes and a small bore needle (<23 gauge) is used. Rapid removal of CSF during the procedure may lead to subdural hematoma or tube blockage due to choroid plexus aspiration, although both are rare. As such, repeat neurologic exams are key following the procedure.

Patients with VP shunt malfunction can sometimes present with many subtle signs and symptoms. Recognizing the historical and physical exam findings in malfunction is essential for the ultimate diagnosis and treatment of a potentially worrisome disease. For the majority of stable patients, tapping a VP shunt should be done by a neurosurgery practitioner due to risk of hardware infection or damage. However, should a patient present with life-threatening symptoms (i.e., Cushing triad), understanding how to complete this skill is vitally important for patient care.

## KEY POINTS

- VP shunts are typically placed to relieve elevated intracranial pressures due to hydrocephalus.
- VP shunt malfunction is due to either an infection or VP shunt component obstruction.
- Neurosurgery should be involved in decision-making when considering VP shunt tap.
- Preferably a neurosurgeon should perform the shunt tap, but in case of emergency, use a 25-gauge butterfly needle to access the port while using sterile procedure.

- Manometry plays a vital role in VP shunt malfunction.

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## **No IV, CONSIDER THE IO**

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Intraosseous (IO) access can serve as a lifesaving alternative to intravenous (IV) access, especially in the crashing hypovolemic patient. The noncollapsible medullary space of bone is a reliable entry point into the vascular system in patients with difficult or delayed IV access.

In the crashing patient, do not let IV access serve as a delay in administering lifesaving medications, fluids, or blood products! Instead, move quickly to obtaining IO access. Numerous studies have shown that IO access is faster, easier, and just as reliable as IV access. This is true for patients of all ages (newborns, infants, children, and adults).

Depending on the clinical scenario, IO access can be placed in the proximal humerus, proximal tibia, or distal tibia. There are no limitations to what can be instilled through the medullary space of the bone. All medications, fluids, and blood products can be transfused through the IO route. However, infusion rates do depend on the site of access. The proximal humerus has been shown to have an infusion rate under pressure of ~5 L/h, compared to ~1 L/h through the proximal/distal tibia.

When placing an IO line it is important to palpate for your landmarks. In the extended leg, the proximal tibia insertion site is ~2 cm below the patella and 2 cm medial. The distal tibia insertion site is located ~3 cm proximal to the most prominent aspect of the medial malleolus. The IO needle should be inserted at a 90-degree angle when accessing the tibia. When accessing the proximal humerus, position the patient with the hand resting on the abdomen (so that the elbow is adducted and the shoulder is internally rotated). The humerus insertion site is on the most prominent aspect of the greater tubercle. The needle should be angled downward at a 45-degree angle.

Once your landmarks have been verified, the needle should be inserted



through the skin and soft tissue until it comes in contact with bone. Do not engage the drill until the needle has passed through the skin/soft tissue and is resting in the appropriate orientation on the bone! At this time, the drill should be engaged with firm pressure until the operator feels a pop as the needle enters the medullary space of the bone. Needle length should be chosen based on the level of overlying soft tissue. If your needle is resting on the periosteum and you are unable to see at least one black line on the needle shaft—your needle is too small and will not be able to properly enter the medullary space!

Basic lab work is vital in ED management decisions, particularly in the critically ill. A CBC drawn from an IO will have hemoglobin and hematocrit levels that closely correlate to IV blood samples; however, platelet level may be low and WBC count may be high. Additionally, many of the lab values from the BMP including glucose, BUN, creatinine, chloride, albumin, and total protein levels will be similar when compared to IV blood draws; however, potassium, CO<sub>2</sub>, sodium, and calcium levels may vary. Blood drawn from the IO line should be sent for analysis; however, it should be interpreted in the correct clinical context!

Contraindications to IO placement include overlying cellulitis/abscess, fracture, and previous or failed IO placement within the past 48 hours in the target bone. A fracture or previous IO placement in the target bone will lead to extravasation of fluid out of the medullary space and into the soft tissue, which places the patient at risk for compartment syndrome!

Removal should be performed within 24 hours. Simply attach a Luer-Lok syringe onto the IO needle and apply firm upward force while turning in a clockwise fashion. Pull straight out; do not bend or rock the needle as this can lead to a retained foreign body!

## KEY POINTS

- Intraosseous access can be obtained in patients of all ages (newborns, infant, children, and adults).
- Common intraosseous access sites include the proximal humerus, proximal tibia, and distal tibia (may remain in place for 24 hours).
- All medications, fluids and blood products can be administered through the intraosseous route.
- Flow rate varies depending on insertion site: ~5 L/h through the humerus and 1 L/h through the tibia.
- IO access is contraindicated if a fracture is present in the targeted

osseous infusion site or if previous IO access has been attempted in the same site in the past 48 hours.

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# **WHAT NERVE! ULTRASOUND- GUIDED REGIONAL NERVE BLOCKS**

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Emergency physicians (EPs) are truly acute pain specialists; many patients who present to the emergency department experience some degree of discomfort that lead them to seek medical care. Pain is best controlled when it is considered on a continuum and approached from a multimodal standpoint. Providers may start with less invasive methods, such as oral analgesics, and escalate to parenteral opioids or procedural sedation to optimize comfort for the patient. In the emergency setting, regional anesthesia in the form of peripheral nerve blockade provides a safe and effective adjunct to systemic analgesia for a large breadth of acutely painful conditions. Whether performed for a complex laceration repair or for additional pain control in a fractured extremity, regional nerve blocks are somewhat underutilized given the convenience and simplicity of the technique. Utilization of regional nerve blocks has been shown in the literature to significantly reduce pain levels and the quantity of required opioid analgesics, particularly if initiated early in patient care.

Historically, nerve blocks have been performed using anatomic landmarks; common applications consist of dental and digital nerve blocks. Up to 30% of nerve blocks fail when the landmark technique is utilized. With the expanding availability of ultrasound and its incorporation into EP training, ultrasound-guided regional anesthesia has become a promising adjunct in emergency pain management. Ultrasound guidance provides real-time needle localization to negate the effects of anatomic variability. It also allows the operator to directly visualize with greater accuracy the

administration of the local anesthetic around the nerve.

Peripheral nerve blockade performed with sonographic guidance has been proven to result in lower volumes of anesthetic administered and faster and more effective anesthesia with fewer complications. Nerve blocks such as the interscalene brachial plexus block can be utilized for anterior shoulder dislocation reductions that would otherwise require procedural sedation. Procedural sedation requires continuous hemodynamic monitoring and also poses serious risks including respiratory depression and hypotension. Emerging literature suggests that regional anesthesia requires less one-on-one provider time, results in fewer complications, and may achieve greater patient satisfaction compared to systemic therapy.

An indication for ultrasound-guided regional anesthesia that is gaining popularity is the use of a femoral nerve block for the management of pain secondary to femur fractures. Its aforementioned benefits become particularly relevant in elderly patients, who constitute over 80% of hip fracture cases in the United States and pose a therapeutic challenge with their heightened tendency toward opioid-induced delirium. Femoral nerve blocks used in this population can help prevent complications due to opioid use, including delirium, aspiration pneumonia, urinary retention leading to urinary tract infections, and prolonged hospital length of stay. While this is one of the simpler peripheral nerve blocks to perform, failure to inject deep to the fascia iliaca is a common reason for femoral nerve block failure.

Many other applications for ultrasound-guided regional anesthesia exist within the emergency department setting. Posterior tibial nerve blocks are an excellent analgesic option for injuries to the plantar surface of the foot, including lacerations, foreign bodies, and calcaneal fractures. Forearm nerve blocks provide adequate anesthesia to the hand, but less so for more proximal injuries such as distal forearm fractures. A brachial plexus block may be helpful in more proximal upper extremity injuries. Providers must be aware that interscalene brachial plexus blocks result in a host of well-known side effects such as phrenic nerve palsy, temporary Horner syndrome, and hoarseness due to recurrent laryngeal nerve palsy. An understanding of the various indications, complications, and pitfalls of various nerve blocks is essential to procedural safety and success.

The choice of local anesthetic, technique, and complications are relatively similar for all nerve blocks. Successful anesthesia requires knowledge of the anatomy of the peripheral nervous system; often, more than one peripheral nerve must be blocked to achieve appropriate analgesia. A thorough assessment and documentation of a neurovascular exam should be performed prior to using any nerve block. Recommendations for technique

include aspirating frequently when advancing the needle and slowly injecting in 3- to 5-mL aliquots. One should withdraw the needle several millimeters if significant resistance is encountered or when the patient feels painful paresthesias, as this may represent intraneural injection.

In choosing which local anesthetic to administer, one must consider the indication for the nerve block, especially with regard to the onset of clinical effects and desired duration of analgesia. Care must be taken to avoid exceeding recommended dosing. It is important to know the signs and symptoms of local anesthetic systemic toxicity (LAST). Within 5 minutes of supratherapeutic injection, LAST will typically first manifest as signs of CNS excitation, such as perioral numbness and agitation. These symptoms can then progress to refractory seizures, coma, and even cardiovascular collapse. While the risks of LAST are extraordinarily low in the setting of direct visualization under ultrasound guidance, any practitioner performing a nerve block should know the unique treatment for LAST, which is intralipid therapy.

Ultrasound guided nerve blocks are frequently utilized by anesthesiologists to facilitate pain control in the critical care and surgical settings. They are also gaining popularity in the contemporary emergency department setting, and their distinct advantages are becoming areas of investigation in emergency medicine literature. As more EPs graduate with significant ultrasound experience during their residency training, ultrasound-guided peripheral nerve blockade should be considered a useful tool in the skilled EP's armamentarium.

## KEY POINTS

- Consider a femoral nerve block for pain control in elderly patients with femur fractures.
- Ultrasound-guided peripheral nerve blockade can be utilized as an adjunct to systemic therapy for the management of acute pain in the emergency department.
- Successful anesthesia requires an appropriate knowledge of the anatomy of the peripheral nervous system; oftentimes, more than one peripheral nerve must be blocked in order to achieve appropriate analgesia.
- A thorough assessment and documentation of a neurovascular exam should be performed prior to any nerve block.
- Prior to performing a nerve block, the practitioner should know the

unique signs and symptoms and the therapy for local anesthetic systemic toxicity.

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## A NEEDLING ISSUE: DECOMPRESSING TENSION PNEUMOTHORAX

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**ARUN NAIR, MD, MPH**

### RECOGNIZE      WHO      NEEDS      NEEDLE DECOMPRESSION

Your increasingly tachycardic respiratory distress patient with distended neck veins, who is now becoming hypotensive should activate your spidey sense for tension pneumothorax. At risk, are your COPDers with blebs, tall and skinny men, bong/pipe hitters, and obviously anyone with chest trauma. It's important to remember not all pneumothoraxes require decompression. Many pneumothorax <10–15% the size of the pleural cavity, can be managed noninvasively and will resorb without further intervention aside from careful monitoring. And those that do require emergent intervention ultimately will need a chest tube. The only specific subset that requires *the needle* are unstable patients, that is, the tension pneumothorax. Be kind to your stable patients—don't poke them with a needle and then be compelled to stuff a tube in a different hole just because there's a pleural edge on the plain film. The needle is only indicated if there's tension physiology with impending respiratory or hemodynamic compromise.

It's also important to remember that just because there's a needle in the chest, that doesn't necessarily mean it's decompressed. All too often, you will get report from EMS that there was a rush of air and symptom improvement only to find that the needle never made it through the chest wall. Or maybe it did, but then, it kinked and the pneumothorax

reaccumulated. Listen to the patient, assess the vitals, and decide if another needle attempt is indicated. A tension pneumothorax should never be diagnosed by CXR!

## LANDMARKS

You've made sure that the patient in front of you is the real deal in terms of symptomatic tension pneumothorax with impending compromise. Then you found the 2nd intercostal space in the midclavicular line and plunged your needle. Seems straightforward enough—what's the catch? First, the space may not be where you think it is: providers often confuse the mammillary line for the midclavicular line as well as fail to appropriately identify the 2nd ICS. These mistakes increase the risk for injury to the internal mammillary, subclavian, and cardiac vessels. Try this technique:

- Find the sternal notch and slide your finger down midline until you feel a bony ridge—this is, the angle of Louis and where the 2nd rib attaches. From here, march your fingers laterally onto the 2nd rib and then inferiorly into the intercostal space below it. Continue to march out in that curved soft space until halfway between the sternal notch and AC joint and this marks the spot for needle entry. Depending on habitus and sex, this site can be anywhere in relation to the nipple.
- As the fattening of the world continues, the chances of that catheter getting into the pleural space even with correct location get slimmer. Some studies suggest that up to 1:2 chests would fail to be decompressed with our standard 45-mm catheters at the 2nd ICS! The pectoralis alone on muscular males can be thicker than this. In these patients, consider placement at the midaxillary line of the 5th ICS (the same location you would place a chest tube) as the chest wall tends to be thinner with less overlying muscle tissue and no major blood vessels. This spot also can be hard to locate on larger patients, so practice this technique: With the patient supine and shoulders relaxed, place your open hand with thumb extended and palm facing toward the patient into his or her axilla until your thumb overlies the deltopectoral groove and then push up as far as you can go without raising the shoulder. Unless you have to buy gloves in the children's department, the breadth of your palm at the 5th MCP should approximate the 5th ICS at the midaxillary line. In the super-morbidly obese or bodybuilder, the chest wall may still be too thick and you should consider use of a longer needle (a spinal needle) or even a quick cut down to the rib before needle placement. Keep in mind that the more overlying tissue there is, the greater the risk for the catheter to kink and for the pneumothorax to



reaccumulate. The other benefit of 5th ICS placement is that this is often where the chest tube will go, which saves the patient from having an extra hole.

## **DON'T FORGET THE US MACHINE!**

While it's said you should never diagnosis a tension pneumothorax on CXR, the few seconds it takes with the probe to differentiate air versus fluid in the chest, see what the heart is doing, and find an intercostal space you can't even feel while someone is grabbing you a catheter seems a worthwhile use of time. Don't forget that US is useful after decompression too, as it can help you track if the pneumo is reaccumulating or see if the needle is still in the pleural space.

## **WHAT ABOUT THE BOUGIE?**

Though not useful in needle decompression, the bougie can be invaluable in chest tube placement. Rather than using your finger to hold open your tract to the pleural space while trying to complete the rest of the tube delivery one-handed, consider using a bougie as your "wire" for a modified Seldinger technique. After entrance into the chest cavity with the Kelly and quick confirmation of the pleural space with a 360 sweep of your finger, advance the bougie cephalad just far enough to ensure that it will not fall out. This will free up both your hands while ensuring you don't lose your tract, just like placing a central line. It can also prevent you from accidentally cutting your finger or losing a piece of glove in the chest cavity on that super-sharp broken rib that's most likely still moving. It also helps to direct delivery of your chest tube cephalad. Keep in mind that the modified Seldinger technique using a standard bougie will only work for larger French chest tubes.

### **KEY POINTS**

- Tension pneumothorax is a clinical diagnosis, NOT a radiological finding.
- Clinical silent pneumothoraxes may not require invasive procedures or can await sterile chest tube placement.
- Needle placement does not guarantee decompression. Know your landmarks for both the 2nd and 5th intercostal spaces as body habitus significantly affects success.

- Ultrasound can be a powerful decision-making tool, and bougies can be useful adjuncts in chest tube placement.

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# **WHICH LINE IS IT? CENTRAL LINE PLACEMENT**

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MD**

Placement of a central venous catheter is a commonly used resuscitative procedure in the emergency department, and the risk of complications cannot be overlooked. This chapter describes novel and evidence-based techniques to avoid the most common complications of central line placement: arterial puncture and subsequent hemorrhage and hematoma. In addition, it will provide guidance into selecting the appropriate anatomical site for line placement in order to avoid site-specific complications.

## **AVOIDING ARTERIAL INJURY**

Arterial puncture and hematoma formation are the most common complications of central line placement. These complications are often a result of dilation and insertion of the catheter into an artery and not puncture with the initial access needle. Therefore, it is critically important to ensure venous cannulation by confirming that the guidewire is passed into the venous system. Two techniques, ultrasound guidance and pressure measurement, have been shown to decrease the rates of arterial injury.

It has been widely demonstrated that two-dimensional (2D) dynamic ultrasound imaging, in which the tip of the needle is visualized in real time entering the intended vein, significantly decreases rates of arterial puncture. Many studies have revealed that arterial placement of the catheter still occurs despite the use of ultrasound needle guidance. Speculated causes include movement of the needle into the artery after removing the ultrasound probe, mistaking the shaft of the needle for the tip, or creating an arteriovenous tract

prior to finding the needle tip in the venous lumen. In order to minimize the risk of arterial cannulation, ultrasound should also be used to confirm guidewire placement within the vein prior to dilation of the vessel.

A second technique to avoid arterial injury is measurement of pressure via the needle. Studies have shown that almost 1% of arterial punctures were not recognized by color and pulsatile flow of blood from the needle. A large retrospective analysis of over 9,000 central line placements with mandatory use of pressure measurement resulted in zero arterial catheter insertions. To measure pressure, attach sterile tubing or a short plastic catheter (the tubing that contains the guidewire can be used in a pinch) to the needle and hold it vertically while watching for the rise of blood. Blood that continues to rise and overflow from the tubing indicates arterial pressure, whereas blood that ceases to rise or gradually starts to fall back towards the needle indicates venous pressure. There are also commercially available sterile manometers that can be used to confirm venous pressure. It should be noted that either the sterile tubing or manometer can be attached to either a needle hub or the short plastic catheter included in the kit. It is recommended to use the catheter, as manipulation of the tubing while attached to the needle can result in movement of the needle tip into the artery or out of the vein altogether. This method may not be useful in very hypotensive patients, as low arterial pressure may be mistaken for a venous pressure.

In addition to the above techniques, a stat blood gas may be performed prior to cannulation in order to ensure venous access, with the caveat that this technique may be more cumbersome and time intensive.

## **SELECTING THE APPROPRIATE SITE**

Site selection is critical in avoiding complications and optimizing success. The subclavian approach is useful for patients with cervical collars or patients with severe orthopnea who must remain in a sitting position. However, the subclavian vein is not in a compressible site, which limits the ability to apply compression in response to an arterial puncture. Further, the clavicle can decrease the ability to visualize the vein with ultrasound. The subclavian vein can be cannulated from a supraclavicular or infraclavicular approach. Reviews have demonstrated that the supraclavicular approach is less likely to result in iatrogenic pneumothorax and has higher success rates than does the infraclavicular approach.

The internal jugular approach allows superior ultrasound visualization compared to other sites, both in locating the target vein and in demarcating adjacent arteries. Furthermore, this site allows easy compression and visualization of an expanding hematoma. However, access may be difficult

in certain situations such as ongoing chest compressions, complicated airway management, or patients with cervical collars or neck injuries.

The femoral approach is useful in a patient undergoing chest compressions, since the insertion site is located away from the moving chest wall. In addition, there is no risk of iatrogenic pneumothorax and the artery is in a compressible site. However, the long-term risk of catheter-associated deep venous thrombosis is significantly higher in femoral lines, and the rate of catheter-associated blood infections may also be higher in femoral lines, although the data surrounding this issue have provided mixed results.

## CONCLUSION

Minimizing complications of central line placement is essential to the emergency physician. Careful consideration of site selection prior to line placement will help minimize immediate and delayed complications. In addition, ultrasound visualization of the needle and guidewire in the venous lumen combined with manometric confirmation of venous access will reduce arterial injuries.

## KEY POINTS

- Maximize success rate of venous cannulation by using real-time 2D ultrasound to visualize needle cannulation of desired vein
- Use ultrasound to confirm wire placement throughout as much length of vein as possible prior to dilation and catheter insertion to avoid dilation of an artery
- Use pressure testing with sterile tubing or digital manometry to confirm venous placement, but not in extremely hypotensive patients
- Use the supraclavicular approach for subclavian lines to minimize risk of iatrogenic pneumothorax
- Consider patient anatomy, clinical condition, and site-specific risks to select the most appropriate approach for central line insertion.

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# **SIZE MATTERS; SPONTANEOUS PNEUMOTHORAX: CHEST TUBE VERSUS PIGTAIL**

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**DERRICK ASHONG, MD**

Chest tubes have served as the main modality for pleural evacuation since they became popular (and the standard of practice) near the time of the Vietnam War. The evolution in its components and indications has served to assist in better outcomes for patients with fewer complications. As with many medical advancements, further time and research have led to new innovations such as the pigtail catheter. With the increased push for limiting hospital admissions, tools like small bore catheters have increased in popularity due to their known cost savings compared to the standard chest tube. However, with the introduction of pigtail catheters, the advantages and limitations of each modality require further clarity.

By definition, a pneumothorax develops from the accumulation of air in the pleural space due to some mechanism. One of these mechanisms, spontaneous pneumothorax, can be divided into two types: primary and secondary. Primary disease is seen in patients with no underlying lung problems, which tends to manifest in tall, thin young adults. Secondary pneumothorax more commonly occurs in patients with known lung disease, typically in older patients with ailments including chronic obstructive pulmonary disease, prolonged tobacco use, asthma, or interstitial lung disease. Whatever the cause, each of these diseases requires removal of excess air via either standard chest tube or small bore chest tube.

A small bore chest tube is a 16-Fr or smaller chest tube inserted via Seldinger technique typically via a premade kit. Many small bore catheters

curl at their end to prevent drain dislodgement after removal of an insert. This detail is reminiscent of a pig's tail; hence their more colloquial designation as "pigtail catheters." The user applies local anesthesia to either the standard fourth or fifth intercostal space at the midaxillary line or the more rarely used site in the second intercostal space at the midclavicular line using sterile technique. This method is less invasive than the standard tube thoracotomy, which involves blunt dissection of the deep tissue and widening of the pleural hole prior to chest tube insertion.

For both procedures, the indications remain the same for a spontaneous pneumothorax. Using the American College of Chest Physicians definitions, a chest tube should generally be considered in large pneumothoraces. A large pneumothorax is defined as chest x-ray evidence of pneumothorax  $> 3$  cm from apex to cupola. Small pneumothorax in primary and secondary pneumothorax require variable observation periods between the two, but typically do not require chest tube insertion assuming no decompensation or evidence of expansion.

When compared head to head, there are growing data showing the increased use of small bore chest tubes. A recent prospective, randomized study indicates that small bore catheters lead to less insertion site pain on the day of, and two days following, insertion. A retrospective study of 91 patients showed that there was no difference in length of stay, recurrent rates, or complications. In fact, small bore chest tubes were found to have a success rate of 88.7%. Another study also found similar resolution rates (around 5 days) between both modalities with fewer complications. Due to the similar resolution rates, patient's with small bore chest tubes are candidates to be discharged home from the emergency department with close outpatient follow up if the patient is (a) clinically stable and (b) shows evidence of lung reexpansion after repeat chest x-ray and observation (typically 4 to 6 hours). These patients typically require a Heimlich valve (a one-way flutter valve device) at time of discharge. One study looking at spontaneous pneumothorax patients found an average cost of \$926 with small bore chest tube placement (the sum total of two outpatient visits and imaging) compared to the \$4,276 a standard chest tube placement incurs given the need for hospital admission. In this study, unlike others, patients were discharged after at most a 2 hour observation period, and no follow-up imaging was done. A majority of the available literature finds small sample sizes for each study as well as a lack of comparison in critically ill patients.

Complication rates are also similar between the two groups. Both catheters are at risk of causing injury to surrounding structures (liver, diaphragm, spleen, and heart) as well as infection and bleeding. User-associated risks like malposition or bending of the tube are also well-known



complications. Studies looking at the complication risk between these two modalities found no clinically significant difference between large and small bore catheters. Of note, the dislodgement rate of small bore chest tubes in one study was found to be 21%.

While large bore chest tubes have traditionally been indicated in pneumothoraces, small bore chest tubes are a worthwhile alternative in spontaneous pneumothorax. Cases involving mechanically ventilation or medically unstable patients have not been thoroughly explored. Given the increased possibility of air leak, a large bore chest tube should be considered until more data becomes available in these patient populations. Ultimately, the use of small bore chest tubes in medically stable patients is an optimal treatment option for patients with large spontaneous pneumothorax, no matter the type.

## KEY POINTS

- Small bore chest tubes (<14 Fr) are associated with less insertion site pain than are large bore chest tubes (>16 Fr).
- Small bore chest tubes are typically easier to insert using the Seldinger technique.
- When compared head to head, small bore chest tubes have similar complication and effectiveness rates
- Outpatient management with a Heimlich valve and follow-up chest x-ray is a viable option in certain patients with small bore chest tubes.
- Large bore chest tubes are still indicated for critically ill or mechanically ventilated patients.

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**SECTION XXI**

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**PEDIATRICS**

## RECOGNIZE CHILD ABUSE EARLY

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**CLIFFORD C. ELLINGSON, MD**

It is estimated that ~896,000 American children are abused and neglected each year, while child fatalities due to abuse are estimated to be more than 2000 per year. The long-term consequences of child abuse can be devastating to the abused, the abuser, and their community. The overall burden of abuse expands far beyond physical injury. Abuse, in all its forms, can affect a child's psychological health throughout his or her life. However, the full effects of abuse are unmeasurable and unfortunately often under appreciated.

Children who are victims of abuse have a higher number of visits to the emergency department compared to nonabused children. It is estimated that 20% of children who die from abuse were seen by a medical provider within 1 month prior to their death. If physical abuse is not detected at the initial medical evaluation, there is a 50% chance for recurrent abuse and an astonishing 10% chance of death. Hence, the emergency department is a critical place for identification and prevention of abuse and its subsequent mortality. Fortunately child abuse can often be recognized through a careful history, a thorough physical exam, and by maintaining a dutiful level of suspicion.

Abuse can be first suspected after gathering a history. Historical information that should raise suspicions for abuse includes the inability of a caregiver to explain a significant injury. Internal alarms should further sound when there are changes in significant details, or the history is inconsistent with the physical exam findings or with a child's developmental capabilities. Don't simply blow off a suspicion for abuse. A history that doesn't make sense is a history that needs further exploration and should prompt a meticulous physical exam. This includes but is not limited to a complete skin assessment of the head, neck, extremities, buttocks, genitals, and back. The oral cavity should be visualized for oral trauma and the extremities, trunk,

and chest wall palpated for boney deformities and pain.

Bruising is common in the ambulatory child who often have bruises along their shins, posterior elbows, and forehead. However, the likelihood of having a noninflicted bruise in a preambulatory child is <1%. Maltreatment should be considered in infants presenting with bruising who have not yet started cruising (around 9 months of age). Bruises seen in areas of the body such as the buttocks, back, trunk, genitalia, inner thighs, cheeks, earlobes, neck, or philtrum are not typical for normal childhood activities. Pattern bruising results from instruments such as belts, cords, shoes, hands, bites, and household objects. These markings can have revealing patterns and always should raise a concern abuse.

Burns comprise ~6% to 20% of all child abuse cases. The most common mechanism is a scald burn from hot tap water. Burns concerning for abuse include burns with symmetric distribution, that are crease sparing, or well demarcated in a stocking or glove pattern suggesting immersion. Additionally burns to the buttock, posterior neck or back, in multiple locations, or requiring intensive care management should be thoroughly investigated for abuse.

Abusive head trauma is a significant cause of morbidity and mortality in abused infants. Abusive head trauma can be easily misdiagnosed, and practitioners should have a low threshold to obtain CT head imaging in the suspected abused neonate or nonverbal child. Subdural hemorrhages are a common intracranial injury associated with abuse and should prompt admission for a complete inpatient abuse workup. Subdural bleeds, which are bilateral, have multiple radiographic densities suggesting repeated injury, or in an intrahemispheric location are particularly specific for abusive head trauma.

Blunt abdominal injury is a rare but highly fatal form of child abuse. Only 1% of children hospitalized because of abuse sustain abdominal injury. Don't depend on abdominal bruising, as it is often not seen, even with severe blows to the abdomen, to determine if imaging is necessary. Liver and pancreatic enzyme test and a urinalysis can be helpful in screening children for abdominal trauma. Imaging is warranted in cases when there is a history of abdominal trauma, abnormal abdominal exam, or abnormal laboratory data. Imaging should also be utilized when the physical examination is unreliable because of patient age, complicating injuries, or mental status changes.

Skeletal injuries can often times be difficult to detect in the nonverbal patient. If concerns of inflicted injury are suspected after a history and physical exam, radiographic imaging should be obtained. If fractures are

discovered in a nonverbal child, particularly in children younger than 2 years of age, then an entire high-quality skeletal survey should follow. Don't just limit imaging to the areas of concern. The goal is to not only identify painful acute injuries but also older nonpainful injuries. A verbal child, who can reliably express pain, should have all painful areas imaged but still may benefit from an entire skeletal survey to again evaluate for any old fractures. Abuse should be considered in all nonambulatory children who present with a fracture. Fractures that are more consistently with an abusive mechanism include metaphyseal injuries (bucket handle or avulsion fractures), fractures of the scapula, posterior rib, spinous process, and sternum. Additionally, multiple fractures, especially of differing ages, are concerning for abuse. Skull fractures concerning for abuse are more often bilateral, multiple, and cross suture lines.

Physicians have a legal and ethical responsibility to report any suspicions of abuse. When abuse is suspected, social worker consultation can help facilitate involvement of the appropriate agencies. If a child abuse specialist is available, early consultation should be placed. If the abused child cannot be safely discharged or the extent of the abuse appears broad, then admission is warranted.

## KEY POINTS

- History and a physical exam are essential.
- If fractures are discovered or suspect in children younger than 2 years of age with concern for abuse, then an entire high quality skeletal should follow.
- Don't depend on external findings when considering abdominal imaging in suspected abuse.
- Concerns for abuse should be reported and investigative agencies contacted.

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## TIPS FOR MANAGING ALL THAT IS PEDIATRIC RESUSCITATION

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**JASON SAUNDERS, MD AND HEATHER SAAVEDRA, MD**

Respiratory arrest, 5 minutes by ground, emergency medical services (EMS) can't secure an airway. As an emergency medicine provider, this is something we come across nearly every shift, except for one thing, this patient is a child. So what makes this so different you ask? For starters, children are not just little adults. All arrests, all resuscitations, are stressful, but this will be on another level. You will likely have a large audience including parents. So how do we as providers ensure that this goes smoothly? In the following pages are some suggestions, or guidelines, to help everyone through the challenge of pediatric resuscitation.<sup>1</sup>

- 1) First, know "normal."
- (a) Quick, what's the normal heart rate range of a 9-week-old? How about a 9-year-old? We are all good at assessing sick or not sick from the door in adults. However, children have a remarkable ability to look great, until they don't, and then you're out of time. Pediatrics is from infancy to adulthood and everything between. Aside from spending hours doing well child checks in clinic, how do we as adult providers expect to know all of this? Pocket cards, phone applications, the Internet, or even a textbook, whatever you're comfortable with, take 2 seconds before going in the room to remind yourself what is normal for this age patient.
- (b) Always be able to identify and explain abnormal vitals in the pediatric population. It's how you'll spot the sick patient before they fall off the edge of the cliff, and it will be a marker to guide a successful resuscitation. Children have much a larger ability to compensate, and if you're seeing



hypotension, you are late and need to act quickly.

2) But my patient can't tell me what's wrong?

- (a) Remember in medical school learning how to do a complete physical exam? Yeah me neither, but brush off the cobwebs and let's take a trip down memory lane. All patients, but especially children, need a thorough physical exam. There's a reason all trauma patients get exposed, so fully expose your pediatric patient. Look for hair tourniquets in the fussy infant. Be cognizant of limb asymmetry to clue you in to fracture. Parents know their child's normal better than you, if they insist something is wrong, take a closer look. Get a good history from EMS before they leave: What did the scene look like? How did they find the patient? etc.
- (b) When your patient can't articulate his or her concerns, and in all likelihood your chart biopsy will be unlikely to yield much history, the physical exam is how you will determine the disposition of the pediatric patient.

3) Don't make the problem worse

- (a) That wheezing, croupy, or stridulous child that is comfortable in mom's arms should stay there. That doesn't mean don't do a thorough exam (see above), but a calm child is a child that will maintain his or her airway much longer. The same goes for your treatments. Use your resources and double check your doses. A dose of epinephrine will be a lot different in that coding neonate compared to a toddler or adolescent.
- (b) Medications, fluid boluses, and even feeding volumes are all weight and aged based in children. If there was ever a time to measure twice and cut once, it is in the pediatric population. Get as accurate a weight as you can, use the Broselow tape, or use age appropriate recommendations in a code, but don't use adult doses ever in children without first double checking. Fluid resuscitation should start with a bolus in a child without a heart history at 20 mL/kg, and you may safely repeat as needed up to three times.

4) Situations to keep in mind

- (a) Ductal-dependent lesions—shocky neonate, don't forget about the Patent Ductus Arteriosus (PDA). Prostaglandins will keep it open. Alprostadil (PGE1) is 0.03 to 0.4 mcg/kg/min, watch for apnea and be prepared to intubate.
- (b) Nonaccidental trauma—have a basic understanding of developmental milestones, 2-week-old infants don't roll off tables and 6-month-old infants don't walk. Don't forget your pathognomonic fractures and exam findings. Torn frenulums, spiral fractures, or posterior rib fractures and bruises in the TEN-4 pattern (thorax, ears, neck—or any bruising in a child

<4 months<sup>2,3</sup>) should all send up red flags.

(c) Febrile with a rash—don't forget to ask about vaccine status. Antivaccine sentiment is on the rise, and you may diagnosis measles or varicella.

#### 5) Final thoughts

(a) The Broselow tape is your friend—Know exactly where it is in your department, *before* you need it.

(b) The IO is a great tool, use it early and often when access is difficult.<sup>4</sup> No more than 3 sticks or 90 seconds. Humeral head or proximal/medial tibia for access.

(c) Be aggressive with asthma—Epi and NIPPV work wonders to fend off intubation. On that note, don't forget a wheeze isn't always asthma, keep foreign body in your differential.

(d) Parents should be at bedside whenever possible, including codes. You want them to see that you are doing everything you can for their child.

## KEY POINTS

- Vitals are vital.
- Know how to efficiently utilize your resources.
- Use IO and NG<sup>5</sup> when you're struggling to gain access.
- A complete physical exam is paramount.
- "Normal" varies dramatically by age in the pediatric population.

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# **KEEP THE BABY WARM! AND OTHER STEPS IN NEONATAL RESUSCITATION**

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**ASHLEY GRIGSBY, DO AND JESSICA KANIS, MD**

The delivery of a newborn in the emergency department (ED) is never a planned event and can be wrought with chaos. Current neonatal resuscitation guidelines target situations in a labor and delivery unit with reliable equipment, specially trained staff, and a controlled environment. However, EDs do not have sufficient access to specialized equipment, nor does the staff working in them encounter neonatal resuscitation daily. Because of this, resuscitating a newborn in the ED has its own set of challenges, and emergency providers need to be prepared to handle these situations.

## **BASIC RESUSCITATION**

Approximately 10% of newborns will require basic resuscitation after birth, and providers should prepare for this when a delivery occurs.<sup>1</sup> The first step is to gather basic supplies including warm blankets, a radiant warmer, a bulb suction, a neonatal sized bag-valve mask, and an oxygen source. At this point, it may be helpful to call your hospital's delivery or NICU team.

Once the child is born, ask yourself three questions: Is the baby term gestation? Is the baby crying? Do they have good tone?<sup>1</sup> If the answer to all these questions is "yes," then baby can stay with mom, placed skin-to-skin, and dried and warmed on the mother's chest. If the answer to any of these questions is "no," the baby should be brought to the warmer for further resuscitation.<sup>1</sup> If the gestational age is in question, a general rule is that term

infants will have wrinkles across the entire plantar surface of the foot, rugae on the scrotum, or touching labia majora.

Initial resuscitation consists of warming, drying, and stimulating the baby using warm blankets, which will also stimulate the baby to cry. If the baby is not crying, bulb suction the oropharynx and the nares. Heart rate (HR) can be checked by listening with a stethoscope or by palpating the umbilical cord at the stump. A normal HR is >100 beats per minute (bpm). These initial steps should take no longer than 60 seconds. By 1 minute of life, if the baby is gasping or not crying, or if the HR is <100 bpm, positive pressure ventilation (PPV) should be initiated after suctioning the oropharynx and nares.<sup>1</sup>

Once PPV has been initiated, apply an O<sub>2</sub> monitor to the right hand. Do not be shocked by an oxygen saturation in the 70s! A normal O<sub>2</sub> saturation will be not be present until ~10 minutes of life.<sup>2</sup> Breaths should be delivered at 40 to 60 breaths per minute with an inflation pressure of 20 cm H<sub>2</sub>O. For term infants, FiO<sub>2</sub> should start at 0.21 and 0.40 for preterm infants.<sup>1,2</sup> Remember that 100% oxygen can be toxic for newborns and is reserved for difficult resuscitations. PPV should be provided until the baby is initiating their own breaths and the HR is >100 bpm.

These steps will often result in clinical improvement, evident by an improved HR, tone, and respiratory effort. If not, the baby will require more advanced resuscitation.

## **IMPORTANCE OF WARMING THE BABY**

Warming a baby after delivery is one of the most important steps in resuscitation, but it can be difficult given the rapid nature of ED deliveries. It is recommended to keep a delivery room temperature at 26°C (78°F), which is not a reasonable expectation for an ED.<sup>1,3</sup> A cold baby can be more difficult to resuscitate, and initial measures may not be successful in the setting of hypothermia (<36.5°C). Fortunately, there are many practical ways to accomplish warming infants in a limited resource setting.

Every ED should have a radiant warmer available, and it should be turned on in preparation as soon as you expect a delivery. Warm blankets should be available immediately after delivery and used to warm and dry the infant.<sup>4</sup> Placing the child skin-to-skin with the mother for the resuscitation is a technique often used in third world countries as primary means of warming and can be beneficial in an ED setting.

Premature infants can be even more difficult to keep warm due to their immature body systems and increased heat losses. Infants 28 weeks or less, or weighing <1,500 g (~3.5 pounds), should receive additional means of warmth by immediately being placed in a plastic wrapping.<sup>3</sup> The special plastic bags for infants are not readily available in the ED, but items that can be used instead are biohazard bags, large resealable plastic bags, and food grade plastic wrapping.<sup>2</sup> The baby should be wrapped immediately after birth and covered entirely from the neck down in the plastic wrapping; additional resuscitation steps can be performed with this on the child.<sup>2,4</sup>

## ADVANCED RESUSCITATION

Around 1% of births will require advanced resuscitation. If effective PPV with supplemental oxygen has been provided for more than 30 seconds, and the HR is <60, start chest compressions.<sup>1</sup> Chest compressions should be delivered with a ratio of 3:1 coordinated with breaths, with a target of 90 chest compressions per minute. If giving chest compressions, 100% FiO<sub>2</sub> should be used and intubation be considered. If the infant's HR remains <60 bpm after adequate PPV and chest compressions, epinephrine and volume expansion may be required.<sup>1</sup> However, this requires IV or intraosseous access, which can be difficult in the ED.<sup>2</sup> Advanced resuscitation is a rare event in the ED, and as such, providers will have less exposure to these clinical situations and should review the skills needed.

### KEY POINTS

- Ask yourself three questions: Is the baby term? Is the baby crying? Do they have good tone?
- If the answer to any is “no,” start your basic neonatal resuscitation with warming, drying, and stimulating the infant to cry.
- Keep the baby warm! Use warm blankets, radiant warmers, mother's chest, or plastic wrapping for very small or premature infants.
- In the initial resuscitation of a term infant, oxygen concentration of 21% is recommended—use room air!
- Be familiar with advanced techniques as it is a rare event in the ED.

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## **THE PEDIATRIC AIRWAY: LEARN IT, LIVE IT, CONTROL IT!**

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**GARRETT S. PACHECO, MD**

Emergency physicians (EPs) are infrequently called upon to manage the pediatric airway, but it is imperative that they be prepared to do so in the critically ill or injured child, rapidly and without complication. There are several anatomic and physiologic characteristics that are unique to children that must be considered when managing the pediatric airway. Respiratory function differences make them prone to oxyhemoglobin desaturation, which can be exacerbated in the physiologically compromised critically ill child. Children in shock states are also at high risk for peri-intubation hypotension that can ultimately lead to cardiovascular collapse.

Children <2 years old have different characteristics to keep in mind when preparing for endotracheal intubation (ETI). They have relatively large tongues, a large occiput, and a more cephalad larynx at the C2-C3 level. Their airway is cone shaped just below the glottis, whereas an adult has a cylindrical-shaped airway below the glottis. Their airway is narrowest at the cricothyroid ring, and the epiglottis is soft. The lower airways are also smaller and less developed than those of an older child or an adult. Each of these anatomic features should be taken into account during ETI preparation.

Appropriate tube size is imperative to avoid issues with air leak and ventilation once the airway is secure. Endotracheal cuff pressures should target 20 to 30 cm H<sub>2</sub>O allowing adequate tracheal mucosa perfusion in all ages. For term infants up to 1 year old, the tracheal tube size is typically 3.0 to 3.5 mm. Children 1 to 2 years old typically receive a 4.0-mm tube. The pediatric advanced life support guidelines provide the following formula to obtain the appropriate sized tube for children >2 years old: Cuffed tracheal tube (ID mm) = Age (years)/4 + 3.5.



Children have a lower functional reserve capacity and increased oxygen metabolism. This makes infants and children at higher risk for respiratory fatigue and oxygen desaturation than adults. These physiologic characteristics highlight why it is so important to adequately preoxygenate the critically ill child. Anesthesia literature has demonstrated that healthy children have half the “safe apnea time” than an adult, which is likely even less in the critically ill child.

In states of hypoxemia with intrapulmonary shunt or low oxygen venous saturation, which is seen in pneumonia, atelectasis, or severe sepsis, it is crucial to have adequate preoxygenation. Critical hypoxemia must be avoided as it can lead to seizures, bradycardia, and ultimately cardiovascular collapse. The surviving sepsis guidelines for pediatrics recommend high-flow nasal cannula oxygenation (HFNC) or nasopharyngeal continuous positive airway pressure (CPAP) for respiratory distress and hypoxemia. HFNC helps with oxygenation and has been shown to decrease dead space and even provide some positive end-expiratory pressure (PEEP) of 3 to 5 cm H<sub>2</sub>O. Noninvasive ventilation (NIV) aids with intrapulmonary shunt and recruitment of functioning alveoli-capillary units able to participate in oxygenation-ventilation. These methods could be used for preoxygenation to increase the “safe apnea time” prior to intubation. Apneic oxygenation has not been well studied in the pediatric population. However, considering children’s high incidence of oxyhemoglobin desaturation during the peri-intubation period, it would be reasonable to implement this process in an attempt to avoid or minimize complications.

Peri-intubation hypotension is well documented in the adult literature and has a reported incidence as high as 21% in pediatric emergency department intubations. Children are very sensitive to changes in hemodynamics during the peri-intubation period and are at risk of vagal-induced bradycardia, which may potentiate cardiovascular collapse. The available evidence does not support the routine use of atropine for premedication in emergency intubation. However, in some circumstances, the young child undergoing emergency intubation may benefit from blocking the vagal-induced bradycardia with administration of atropine 0.02 mg/kg (no minimum dose) prior to induction to prevent this complication.

The hypotensive child should be adequately resuscitated prior to any intubation attempt. Intubation and mechanical ventilation increase the intrathoracic pressure, which directly increases right atrial pressure. The increase in right atrial pressure along with the decrease in mean systemic pressure has the effect of decreased venous return. This leads to a decrease in preload, which results in cardiac depression and hypotension. The sedative-

hypnotic agent used for induction may also cause a decrease in blood pressure through sympatholysis. These hemodynamic effects are why the surviving sepsis guidelines recommend volume loading the septic shock patient aggressively prior to an intubation attempt.

As previously mentioned, choice of induction agent can help preclude episodes of hypotension following intubation. Ketamine is an agent that provides analgesia, amnesia, bronchodilation, and preservation of spontaneous respiration and has sympathomimetic properties increasing heart rate and blood pressure. Etomidate is another agent that is reported to be “hemodynamically neutral.” Both agents have their respective drawbacks, and the EP should consider these prior to choosing an RSI agent. The critically ill patient that has depletion of catecholamines may actually have myocardial depression with the addition of ketamine. Etomidate is not FDA approved for use in children less than 10 years old. Also, there are reasonable concerns that etomidate use can lead to increased mortality in children because of its adrenal suppression effect, and it should be avoided in an obviously septic patient.

## KEY POINTS

- The pediatric airway has anatomical differences that if addressed appropriately allow for improved first pass success at intubation.
- The critically ill child has little physiologic reserve. The most experienced intubator should be called upon to manage these patients to avoid potential peri-intubation complications.
- Children desaturate faster than adults. Critically ill pediatric patients will desaturate even faster. Adequately preoxygenate and use apneic oxygenation to prolong the safe apnea time.
- Address hemodynamics prior to intubation to avoid cardiovascular collapse. This is done with adequate volume resuscitation, thoughtful pretreatment and induction choice, and potentially peripheral vasopressors.

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## ALL THAT BARKS IS NOT CROUP

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**SHERYL YANGER, MD**

Infants and children frequently present to the emergency department with stridor. Like many potentially emergency conditions, this often happens in the early hours of the morning when limited resources or specialists are available. It is important for the emergency department (ED) physician to differentiate croup from other chronic or more life-threatening causes of stridor. A careful history and physical examination are necessary to assess whether immediate intervention is required, to determine what might be causing the stridor, and to decide if referral to otolaryngology or other specialists is needed.

As with any patient in the ED, initial exam should focus on determining whether the patient has impending airway collapse. Presence of increased respiratory effort with significant intercostal or suprasternal retractions is more indicative of distress than the degree of stridor. Hypoxia, hypercarbia, or mental status changes signify impending respiratory failure and requires immediate intervention. Of note, do not rely on lab tests to guide your management—look at the patient in front of you and act appropriately.<sup>1</sup> For acute stridor, if the patient's airway appears stable and croup is suspected, a dose of oral steroids may be given. If the child has stridor at rest, a trial of inhaled racemic epinephrine may help alleviate inflammation in the airway. Be aware that the breathing treatment may increase agitation and worsen stridor in some cranky toddlers, so don't be surprised and give the patient some time! Further steps in management depend on the acuity of the symptoms and further findings on exam and history.

After the airway and breathing, focus on associated symptoms and any external indication that there may be an anatomic cause of the stridor. Half of patients with subglottic hemangiomas will have cutaneous hemangiomas, particularly in the "beard distribution." Many syndromes are linked to

congenital airway anomalies such as Down syndrome with subglottic stenosis or CHARGE syndrome with cranial nerve palsy.<sup>2</sup> Careful inspection may reveal lymphatic malformations or facial, neck, or chest masses, which may externally compress the airway. In short, beware of the child with known or suspected congenital abnormalities; if something looks funny in the child, it might extend to the airway.

It is important to determine whether stridor is acute or chronic and congenital or acquired. Ask the parents' details about onset of the stridor, severity, progression, cyanotic or apneic episodes, or retractions. Additional information about birth history, relationship with feeding and body position, suspicion of foreign body aspiration or ingestion, voice quality, reflux, feeding difficulties, aspiration, or any history of pulmonary or neurologic disease is all helpful.

The most common congenital causes of stridor in infants and children are laryngomalacia, vocal cord paralysis, congenital subglottic stenosis, tracheomalacia, subglottic hemangioma, laryngeal webs, and posterior laryngeal clefts. Acquired causes may be traumatic intubation/subglottic stenosis, foreign body, infection, or juvenile laryngeal papillomatosis.

Croup, or laryngotracheobronchitis, is the most common cause of acute stridor in young children and toddlers. It is usually caused by a viral infection of the larynx and upper trachea. A “barking” cough and upper respiratory infection in children 6 to 36 months are pathognomonic. Bacterial tracheitis presents as a toxic-appearing child with cough and progressive stridor. Epiglottitis, the incidence of which has dramatically declined since the introduction of the *Haemophilus influenzae* vaccine, may present as an ill-appearing child with muffled voice, drooling, and tripod positioning. Early consideration of intubation in a controlled setting such as the operating room in these children is recommended.<sup>3</sup>

Foreign body ingestion or aspiration should always be a consideration in children presenting with stridor, especially in the absence of fever or upper respiratory infection.

Laryngomalacia is abnormally floppy supralaryngeal tissue that passively collapses during inspiration. It is typically stable inspiratory stridor presenting in the first 2 weeks of life, worsens in the supine position, and can be aggravated by sleep, feeding, or irritability. Most spontaneously resolve by 12 to 24 months and require no intervention; however, up to 20% may have severe disease that requires surgery.<sup>2</sup>

Bilateral vocal cord paralysis is less common and may be congenital or acquired. It can be present at birth or may take months to present. This

generally requires intervention to secure the airway and tracheostomy while awaiting recovery of neurologic function. It may be caused by Chiari malformation, and these infants require further workup including brain MRI.

Although not necessary as a part of the routine evaluation of a child with typical croup symptoms, plain films of the chest or neck may be useful to visualize radiopaque foreign bodies or air trapping. If symptoms are atypical, imaging may be useful in diagnosing croup or subglottic stenosis (steeple sign), epiglottitis, subglottic masses, tracheal stenosis, or rings. Less commonly, CT or MRI may be used to evaluate the presence of neck or chest masses, or vascular rings or slings.

Chronic stridor, recurrent croup, or progressive symptoms require referral to an ear-nose and throat specialist for fiberoptic laryngoscopy.<sup>1</sup> History of cyanosis, retractions, apneic spells, or any respiratory distress on exam warrant inpatient admission with appropriate monitoring and urgent consultation with a specialist after initial stabilization. A history of pulmonary disease, aspiration, or reflux may warrant follow-up with pulmonology or gastroenterology as well.

## KEY POINTS

- The most common cause of acute stridor is croup, and the second is foreign body ingestion/aspiration.
- A careful history is necessary to distinguish acute or chronic, acquired or congenital causes of stridor.
- Physical exam may give clues to underlying anatomic abnormalities.
- Recurrent episodes of stridor, progressive symptoms, cyanosis, apnea, or respiratory distress all require further evaluation and referral to otolaryngology for fiberoptic laryngoscopy.

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# **DON'T GET IN HOT WATER BY NOT KNOWING HOW TO TREAT PEDIATRIC BURNS**

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The assessment of pediatric burns begins with a “doorway” impression: if the injuries appear severe from the doorway, you will need to start with standard trauma algorithm of airway, breathing, circulation, disability, and exposure. When assessing the airway, examine the inside and outside of the mouth and nose looking for soot, blisters, or burns as these are signs that the patient may have further injury to his or her internal airway. If you have concerns about the patient’s airway, you need to stop and address it immediately.<sup>1</sup> Injured mucosal tissue will swell, making securing the airway much more difficult. When addressing circulation, make sure to palpate pulses in all extremities distal to burn injuries. A circumferential injury may compromise the circulation to areas distal to the injury. Likewise, a circumferential injury to the chest can cause a restriction on breathing and circulation. Get the patient completely undressed as the clothing may be contaminated, be smoldering, or hide additional injuries. As always, if you encounter a positive finding during your algorithm, stop and fix it.

Once you have the initial life threats addressed, begin the more detailed assessment phase to estimate burn severity, the body surface area impacted by the burns, and the mechanism of the burn injury. Burns are now classified into superficial (epidermis injured), partial thickness (epidermis injured with partial injury to the dermis), and full thickness (epidermis and dermis both with complete injury).

In children, estimating body surface area can be difficult. In general, the palm of the child's hand is considered 1% of the body surface area and can be used to quickly estimate total body surface area impacted. In addition, the modified Wallace Rule of 9s can be applied<sup>1</sup> assigning each area of the body a percentage of the total body surface area. The head (front and back combined) is 18%, front of torso is 18%, back of torso is 18%, each arm is 9%, each leg is 13.5%, and the perineum is 1%. In general, these pediatric parameters are used for any patient who is younger than 5 years old but may be used through puberty.

Lastly, the mechanism of the burn needs to be identified as it will determine specific treatments required for that burn type. Types of burns include electrical, chemical, or thermal. Electrical burns frequently have internal injuries to nerve and muscle that are far more significant than external injuries. Arrhythmias and rhabdomyolysis may result. Chemical burns will often require decontamination of the burn site; therefore, it is important to determine the causative agent, if possible. Scald and contact burns are common in children due to direct contact with hot surfaces or liquids. Make sure the pattern appears consistent with the history and stated mechanism. If there are inconsistencies in the story or a concerning pattern distribution of the burns, remember to consider nonaccidental trauma.

In burn care, there are several immediate factors that need to be addressed. The first is fluid resuscitation and should be initiated immediately in any patient that did not pass the "doorway" assessment. Establishing the correct fluid resuscitation is key because over resuscitation can lead to tissue edema and compartment syndromes where under resuscitation can lead to organ hypoperfusion. The most frequently used formula is the Parkland formula: intravenous fluid volume (in milliliters) [body weight (in kilograms)  $\times$  percentage of TBSA burn  $\times$  4 mL of lactated ringer solution].<sup>2</sup> Fluid for first 24 hours =  $(4 \times \text{kg} \times \% \text{TBSA burn})$ . The Parkland formula accounts for the amount of fluids that need to be given for the burn injury within the first 24 hours with 50% being given in the first 8 hours and the other 50% being given over the following 16 hours. Remember, the clock starts at the time of the injury, not at the time of presentation to the emergency department. Be sure to subtract any fluids that were already given by EMS or an outside hospital from the total, and add in hourly maintenance fluids. The patient's response to the fluid resuscitation is best measured by the placement of a Foley catheter to monitor urine output. The goal urinary output in pediatric patients is 1 to 1.5 mL/kg/h. Early initiation of fluid resuscitation is paramount and needs to be started before the patient is transferred to a burn center. The second factor that needs to be addressed is the patient's pain control. Do not forget these are often very painful injuries



and need to be treated accordingly. If the burn is not painful, then a full-thickness burn may be present.

Once the initial assessment is completed, you will need to contact your regional pediatric burn center to discuss the case. When calling, have your detailed assessment information available including the estimate of burn severity, the body surface area impacted by the burns, and the mechanism of the burn injury.<sup>3</sup> The burn center will provide guidance on transfer and even outpatient follow-up options for more minor burns.

## KEY POINTS

- As in all trauma situations, begin with ABCDEs first. Secure the airway early.
- Use the modified Rule of 9s to estimate the total body surface area and plug that into the Parkland formula to calculate 24-hour fluid resuscitation requirements. Add maintenance! Subtract total fluids given prior to arrival!
- Remember the 24-hour clock for fluids starts at the time of the injury, not at the time of presentation to the emergency department.
- Pain control!
- If you are not at a burn center, contact your regional burn center early for arrangement of transfer and recommendations.

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## MY BABY WON'T STOP CRYING!

**J. ADAM HAWKINS, DO AND TIMOTHY RUTTAN, MD**

Much to the frustration of most physicians, ED providers are frequently confronted with an infant with persistent crying. This seems to occur far more frequently at three in the morning when the parents are at their breaking point of frustration and concern. It is critical for the ED physician to have a systematic approach to these children to avoid a perfect storm of anxiety and frustration on the part of the parents and the potential for significant missed pathology in the infant.

An understanding of normal crying is important to recognize what is potentially worrisome. Crying typically begins shortly after birth and peaks at ~3 hours per day at 6 to 8 weeks of age, and then rapidly decreases until finally leveling off around 4 months of age. Note that it is normal for a young infant to cry for 3 hours a day—not fun for anyone in the household. Proper counseling on what is normal not only helps with clinical decision-making but can also help ease parental anxiety and even prevent future unnecessary ED visits. In addition to the amount of crying, what is unusual is crying that is persistent despite appropriate soothing methods and the passage of time.

Crying can be the primary or sole manifestation of many life-threatening conditions that must be considered in the young infant. Many of these can be excluded by a good history and physical exam, but it is helpful to use a mnemonic to ensure that nothing is missed in a systemic approach. The IT CRIESS mnemonic developed by Herman et al. is a useful device:

- I—Infections (viral infections, urinary tract infection, meningitis, osteomyelitis, and the like...)
- T—Trauma (accidental and nonaccidental), testicular torsion
- C—Cardiac (congestive heart failure, supraventricular tachycardia, myocardial infarction)
- R—Reflux, reactions to medications, reactions to formulas

- I—Immunizations (recent diphtheria-tetanus-pertussis vaccine), insect bites (scorpions)
- E—Eye (corneal abrasions, ocular foreign bodies, glaucoma)
- S—Surgical (volvulus, intussusception, inguinal hernia)
- (S)—Strangulation (hair/fiber tourniquet)

Studies have shown that lab tests are seldom necessary in the evaluation of inconsolable crying, and the history and physical exam remains the cornerstone in the ED. In addition to the standard elements, the directed history should include birth history, recent illnesses or vaccinations, and any suggestive social history or environmental exposures. A thorough and systematic head-to-toe exam should be performed with the patient fully exposed. This means a completely naked infant—hair tourniquets or other important skin findings are easily overlooked by providers who are too worried about the infant’s “comfort” to do the job correctly. One note of caution on the eye exam—corneal abrasions are common in this age group, and some studies suggest that they may be an incidental finding rather than the etiology of the crying. Be cautious about diagnostic anchoring, and be sure to complete your full exam. This is especially the case if the application of topical ocular anesthesia does not relieve the crying.

The real problem becomes what to do after your thorough history and physical fail to provide an answer. A stepwise progression is helpful here in the well-appearing and nontoxic child. Most infants will stop crying by the time the initial evaluation is complete; these are unlikely to have significant underlying pathology and can usually be safely discharged with close outpatient follow-up. If, however, the infant is still crying enough that you can’t stand to be in the room, a stepwise approach including the use of lab, imaging studies, and admission for observation may be in order. Returning to the IT CRIESS mnemonic, these studies may include blood, urine, CSF, and imaging of the abdomen and head to exclude life-threatening pathology in the truly irritable infant. Once again, however, this is a rare phenomenon and should not be a routine part of the evaluation.

One word of caution on the diagnosis of colic, which is likely overdiagnosed in the ED and should rarely be diagnosed on the first patient encounter. Colic is often defined as crying for more than 3 hours a day for at least 3 days a week for more than 3 weeks in duration—the so-called rule of threes. Most important, other pathology must be ruled out over time. As can be seen, this is usually not a one-time snapshot diagnosis, but rather requires observation over time. For the ED provider, it is appropriate to raise the issue of potential colic and discuss what this might mean with the family, but be careful about diagnosing the infant with 24 hours of excessive crying with “colic” out of habit or assumptions.

Perhaps the most important role for the ED provider after life-threatening pathology has been ruled out is to provide support to caregivers. As can be imagined, persistent crying is extremely stressful for caregivers, and the risk of nonaccidental trauma must always be considered. The visit to the ED can provide an opportunity to intervene, counsel, and connect with resources as needed to help avoid future trauma. If available, ED social workers can help connect families with supportive resources, and close follow-up with primary care can help provide ongoing caregiver and patient assistance.

No one enjoys seeing a crying infant, but understanding what is normal and having a systematic approach to the history and physical will help ensure the proper care of these challenging patients and make sure that you aren't reduced to tears of your own.

## KEY POINTS

- Do a complete history and physical in all crying infants, including completely undressing the infant.
- Minimize testing in most patients.
- Give the child some time if the child is well appearing as most crying will stop by the end of the visit.
- Provide support to parents of crying children.
- Remember the IT CRIESS mnemonic to help guide your differential.

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# **PEDIATRIC PROCEDURAL SEDATION IN THE ED: EASIER THAN YOU MAY THINK**

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Bedside procedures necessitating the use of procedural sedation are frequent in emergency departments worldwide. Procedural sedation has proven to be a safe and inexpensive way to facilitate many procedures that would otherwise require general anesthesia or be impeded due to pain and lack of patient cooperation.

Prior to beginning sedation, evaluate the patient's airway and cardiovascular status. There are no absolute contraindications; however, relative contraindications include craniofacial/airway abnormalities, difficult airway, or ASA classification III or higher. In these circumstances, the patient may benefit from sedation in the OR by a pediatric anesthesiologist. Other considerations should include the patient's fasting status, to reduce risk of aspiration. ACEP has issued a level B recommendation to not delay necessary procedural sedation in adults or pediatrics based on fasting time, as duration of fasting has not reduced the risk of emesis or aspiration.

Preparation should include proper monitoring and ensuring all necessary equipment is easily accessible. Monitoring should include ECG/respirations, noninvasive blood pressure, pulse oximetry, and capnography. Capnography is especially useful as a high end-tidal CO<sub>2</sub> or drop-off in respirations will be the first indicator of hypoventilation and impending respiratory compromise. Always be prepared for the worst, and have emergency airway equipment immediately available.

When circumstances permit, sedation should be administered and

managed by a practitioner not involved in performing the procedure. This way, one can focus concentration on patient monitoring, and managing potential complications. Be familiar with the procedure being performed in order to anticipate the amount of time sedation will be needed and the need for additional analgesia during or after the procedure.

Numerous sedative-hypnotics and analgesic agents are available for procedures requiring brief sedation. Choice of agent is largely based on the practitioner's comfort and institutional availability. Commonly used agents include the following:

**Propofol** has gained popularity for procedural sedation. It can be administered as a continuous infusion for longer procedures; however, for short procedures in the ED, bolus dosing is usually sufficient. Propofol is given as a bolus dose of 1 to 2 mg/kg with repeated doses of 0.5 mg/kg as necessary to maintain sedation. Propofol can be combined with another agent such as ketamine or fentanyl—in which case the initial bolus dose should be adjusted to 0.5 mg/kg.

Propofol has the benefit of a very fast onset and a very short recovery time. However, it can be difficult to titrate, which may lead to complications such as respiratory depression and hypotension if not used carefully.

**Midazolam** is a short-acting benzodiazepine that can be given via several routes, which makes it an attractive choice when sedating the pediatric patient. Onset of action is generally fast, but will depend on the route of administration. Midazolam has strong anxiolytic and amnestic properties, but may not have sufficient analgesic properties for some procedures and therefore may need to be combined with an opioid. Respiratory depression may occur, especially if combined with an opioid. Paradoxical reactions are reported in 1% to 3% of children, characterized by aggressive behavior, hyperactivity, and inconsolable crying. These symptoms are self-limited, and if this occurs, another agent should be considered.

**Ketamine** is a very popular choice for procedural sedation in pediatrics. It produces a dissociative, “trance-like” state while preserving respiratory drive, airway muscle tone, and protective airway reflexes. It has a relatively short duration of action, and has great analgesic and amnestic properties. Ketamine can be administered IV or IM. IV dosing is 1 to 1.5 mg/kg with repeated dosing of 0.5 to 1 mg/kg as needed to maintain adequate sedation. IM dosing is 4 to 5 mg/kg with repeat doses of 2 to 4 mg/kg as needed. Note that with IM administration, recovery time may be prolonged. The most frequent complication with using ketamine is nausea and vomiting; thus, many practitioners choose to premedicate with an antiemetic. It is also known to increase airway secretions, and may be a poor choice for certain

airway/respiratory procedures—requiring pretreatment with atropine. Emergence phenomenon may occur causing a brief period of agitation and anxiety.

**Nitrous oxide (N<sub>2</sub>O)** is an inhaled anesthetic gas that provides sedation, analgesia, amnesia and anxiolysis. It has a very rapid onset of action and recovery time, and is well tolerated in pediatric patients. N<sub>2</sub>O gas is administered through a demand-valve mask or continuous flow system. Some systems provide a fixed 50/50 concentration of N<sub>2</sub>O and oxygen, while others will allow the operator to adjust the concentration of N<sub>2</sub>O up to 70%. While it is contraindicated in pregnancy, N<sub>2</sub>O otherwise has an excellent safety profile and adverse effects are minimal—again, due to risk of nausea and vomiting, consider premedicating with an antiemetic.

Of course, sedation is not limited to the medications mentioned above. Other sedatives such as dexmedetomidine, etomidate, and barbiturates can also be used for procedural sedation. It is vitally important to be familiar with the safety profile, duration of action, contraindications, and adverse effects of the agent you're using. Consider the effect of multiple agents being used in conjunction—for instance, use of opiates may reduce the amount of sedative needed to achieve adequate sedation.

Observe the patient in the ED per institutional policy after the sedation until he or she has returned to a baseline mental status and can tolerate PO liquids. Remember to provide additional pain control after discharge as needed.

## KEY POINTS

- Preparation—Always have emergency airway equipment, backup airway equipment, drugs, and suction at bedside. Anticipate anything that can go wrong, and be ready for it.
- Become very familiar with the agent you're using—contraindications, adverse effects, onset of action, recovery time. Keep in mind the additive effects of multiple agents.
- Make sure patient has recovered from sedation and that pain is well controlled prior to discharge.

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## THE INS AND OUTS OF INTUSSUSCEPTION

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Intussusception is a “must make” diagnosis in the emergency department. Failure to quickly recognize and treat this ailment can lead to devastating consequences for your patient up to and including ischemic bowel. Intussusception is a telescoping of the bowel into itself and most often occurs in the ileocolic junction. It is most commonly found in young children under 5 years of age. As the bowel telescopes into itself, it becomes edematous. The mesentery pulls into the overlying bowel loop (the intussusciens), and, in the worst cases, blood flow to the bowel is compromised. Most cases are idiopathic; however, lymphoid hyperplasia (perhaps secondary to an antecedent viral infection) or a previously unknown Meckel diverticulum can induce a lead point.<sup>1</sup>

Clinically, you may see a range of presentations from an intermittently irritable child experiencing bouts of colicky pain as the natural peristalsis of the bowel worsens the telescoping to the other extreme of an apathetic, toxic, listless child. Fits of pain will cause the knees to be drawn up to the chest. A sausage-shaped mass may be palpated in the belly. As the forward flow of GI contents is halted, signs of a bowel obstruction will present themselves. Vomiting (perhaps bilious), dehydration, and even lethargy can be present. The edematous bowel will often lead to third spacing and worsening dehydration. As the bowel mucosa dies and sloughs, the patient can display mucus-laden bloody stool (currant jelly stools). Since the current jelly stools are a late finding, we should aim to make the diagnosis before this occurs. In the most severe cases, the child will experience peritonitis and take on a toxic

appearance.<sup>2</sup>

Early diagnosis of intussusception is key to help prevent ischemic bowel and shock. The initial workup should be ordered in tandem with fluid resuscitation and supportive care in the sick child. Basic labs will help you identify and correct metabolic disturbances. High clinical suspicion is enough to proceed with testing, and the diagnosis is typically made via imaging. Abdominal ultrasound has proven itself to be an excellent test for this purpose. It is quick, noninvasive, and without radiation. It boasts a sensitivity and specificity of 97.9% and 97.8%, respectively, in detection of ileocolic intussusception in the pediatric population.<sup>3</sup> If you find yourself working in a situation where you do not have ultrasound at your disposal, abdominal x-ray may be helpful. A paucity of air, especially in the RUQ should raise your suspicion; however, this test should be used with caution. Paucity of air in the RUQ on 2-view abdominal x-ray was found to have a sensitivity of 62.3 and did not perform well in a recent comparison to ultrasound for the exclusion of intussusception (KUB false-negative rate 37.8% compared to ultrasound 1.6%).<sup>4</sup> Air contrast enema (ACE) has been shown to be safe and effective as the initial treatment for a stable child and can be both diagnostic and therapeutic. Air is the preferred contrast medium over barium or saline (hydrostatic reduction) in any child who does not present with operative indications (bowel perforation, peritonitis, shock, or an obvious pathologic lead point).<sup>5,6</sup> Following diagnosis, early consultation with a pediatric surgeon is advisable as they may wish to be present for the reduction since a bowel perforation can occur (~0.74% of pneumatic and hydrostatic reductions) or if the attempt fails.<sup>5</sup>

The decision to observe for a period of time or discharge following a successful contrast enema reduction remains controversial. Intussusception recurrence rates vary by study; however, a recent meta-analysis reports an overall recurrence rate of 7.5% to 12.7% following contrast enema. This is highest in the first day or two following reduction (2.2% to 3.9% recurrence in 24 hours, 2.7% to 6.6% recurrence in 48 hours).<sup>7</sup> Even though the recurrence rate is low, and serious complications following a successful reduction are rare, the decision to send a child home should only be made after careful discussion with family including reasons to return to the ED. Any evidence of hemodynamic instability, acidosis, inability to return to the emergency department, difficult reduction, or factors placing the patient at a high risk of recurrence may warrant hospital admission. Factors that increase the risk of recurrence include presence of symptoms >24 hours, and location of the intussusception tip at or proximal to the hepatic flexure.<sup>8</sup>

Failure to reduce an intussusception by contrast enema can safely be followed by a second attempt in the well-appearing child. The controversy on this point is dropping as data are surfacing that supports the second attempt. When treating the child with a failed reduction with a second ACE, the NNT to prevent a small bowel resection is 7.<sup>9</sup> These children also experienced a shorter hospital length of stay and decreased hospital costs. Risk factors for failed reduction include symptom duration >24 hours, diarrhea, lethargy, and extent of the intussusception.<sup>10</sup> A second failure will require discussion with a pediatric surgeon for admission and operative evaluation.

## KEY POINTS

- Think of intussusception in any child (especially under 5) who is ill appearing or complains of colicky abdominal pain.
- Evaluate concurrently for dehydration and acidosis as ultrasound is obtained (if available).
- It is reasonable to attempt an ACE on a well-appearing child without evidence of shock, bowel perforation, peritonitis, or pathologic lead point. Refer all others for emergency pediatric surgery consult.
- A second ACE can be attempted in the well-appearing child should the attempt fail, or intussusception recur.
- Discharge home following successful reduction may be reasonable for well-appearing children with the ability to return to the ED, after discussion is had with their parents. Admit all others.

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# DO NOT RELY ON URINALYSIS TO EXCLUDE URINARY TRACT INFECTIONS IN CHILDREN YOUNGER THAN TWO YEARS

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ARTURO S. GASTAÑADUY, MD

Urinary tract infection (UTI) is the most common serious bacterial infection diagnosed in children, accounting for 7% to 14% of pediatric emergency departments (PED) visits. The prevalence of UTI varies with age, sex, race, and circumcision status in boys. For example, UTI prevalence in febrile girls 0 to 12 and 12 to 24 months is about 8% and 2%, respectively. In boys, UTI prevalence is one-third to one-fourth of that in girls; however, the frequency of UTI in uncircumcised boys is 4 to 8 times higher than that in the circumcised boys, reaching 20% in those younger than 3 months. White children have UTI two to four times more frequently than do black children.<sup>1</sup> Other factors associated with increasing prevalence of UTI in young children include urinary tract anomalies, bladder dysfunction, constipation, vesicoureteral reflux, recent urinary tract instrumentation, and family history of UTI.<sup>2</sup>

Most UTI are caused by enteric flora with *Escherichia coli* accounting for about 80% of the cases. Other organisms include *Klebsiella*, *Proteus*, *Enterobacter*, *Citrobacter*, *Staphylococcus saprophyticus*, enterococcus, and *Staphylococcus aureus*. Most of the times, the bacteria reach the urinary tract through the ascending route after colonization of the periurethral mucosa and attachment of the organisms to the uroepithelial cells by glycosphingolipid receptors. The descending (hematogenous) route is also possible especially in

neonates.<sup>2</sup>

Delayed treatment or untreated UTI may lead to extension and/or dissemination of the infection (pyelonephritis, urosepsis) and loss of renal parenchyma (renal scarring) with long-term consequences such as hypertension, decreased renal function, and end-stage renal disease. Consequently, prompt and accurate diagnosis and treatment of UTI are of the utmost importance for affected children.<sup>2</sup>

Current AAP guidelines for the diagnosis and management of initial UTI in febrile children 2 to 24 months of age require “the presence of both pyuria and at least 50,000 colonies per ml of a single uropathogenic organism in an appropriately collected urine specimen.” In this age group, an appropriately collected urine specimen should be obtained by bladder catheterization or suprapubic aspiration (SPA) for urinalysis (UA) and culture before antibiotic treatment. Catheterization is used more frequently. SPA is indicated for males with severe phimosis or females with labial adhesions that preclude access to the urethral meatus. Both methods are comparable; if SPA is used as the gold standard, the sensitivity and specificity of catheterized urine specimens for culture are 95 and 99, respectively. Midstream clean catch urine samples are not feasible in this age group, and bag urine samples give up to 85% false positive cultures and therefore should not be used.<sup>3</sup>

The most used components of the UA for the diagnosis of UTI are leukocyte esterase (LE) and nitrites by dipstick, and the detection of WBC and bacteria by microscopy. LE is produced by neutrophils and suggests pyuria. Nitrites result from the reduction of urinary nitrates by bacteria and indicate bacteriuria. The sensitivities and specificities of individual or combined components of the UA are presented in the table.<sup>3</sup> ([Table 318.1](#))

**TABLE 318.1 SENSITIVITY AND SPECIFICITY OF COMPONENTS OF URINALYSIS ALONE AND IN COMBINATION**

| <b>TEST</b>  | <b>SENSITIVITY<br/>(RANGE) %</b> | <b>SPECIFICITY<br/>(RANGE) %</b> |
|--|----------------------------------|----------------------------------|
| Leukocyte esterase                                     | 83 (67–94)                       | 78 (64–92)                       |
| Nitrite  | 53 (15–82)                       | 98 (90–100)                      |
| Leukocyte esterase or nitrite positive                 | 93 (90–100)                      | 72 (58–91)                       |
| Microscopy: WBC  | 73 (32–100)                      | 81 (45–98)                       |
| Microscopy: Bacteria                                   | 81 (16–99)                       | 83 (11–100)                      |
| Leukocyte esterase, nitrite, or<br>microscopy positive | 99.8 (99–100)                    | 70 (60–92)                       |

Adapted from Subcommittee on Urinary Tract Infection, Steering Committee on Quality Improvement and Management; Roberts KB. Urinary tract infection: Clinical practice guideline for the diagnosis and management of the initial UTI in febrile infants and children 2 to 24 months. *Pediatrics*. 2011;128(3):595–610.<sup>3</sup>

LE is positive in most cases of UTI. False-positive results may occur if the sample is contaminated with periurethral secretions. Other febrile illness (viral cystitis, streptococcal infections, Kawasaki disease) or vigorous exercise may result in sterile pyuria. False-negative LE tests with positive cultures also occur; the most likely explanations are a high cutoff for positivity and diluted urine. Another reason could be asymptomatic bacteriuria, which has been reported in 0.7% of asymptomatic girls 2 to 24 months old.<sup>3</sup>

A positive nitrite test is very specific for the diagnosis of UTI, but it is not very sensitive. False-negative results are common. Urine must stay in the bladder for up to 4 hours to have detectable levels of nitrites, and young children urinate frequently. Furthermore, not all uropathogens reduce nitrates.<sup>3</sup>

Neither over- nor underdiagnosis is acceptable. Concordance of UA and urine culture will confirm or deny the diagnosis of UTI. If, in a catheterized or SPA sample, the UA is positive but the culture is negative, then sterile pyuria and its differential diagnosis should be considered. On the other hand, if the UA is negative and the culture is positive, it is most likely a false-negative UA; UTI remains the most likely diagnosis. Asymptomatic bacteriuria with fever from another cause is another possible explanation.

Culture is indispensable to identify the causal agent of the UTI and its antibiotic sensitivities; therefore, it should be sent for every infant with fever without a source. Furthermore, both SPA and bladder catheterization are invasive procedures, and it would be foolish not to obtain the maximum information from a urine sample obtained by these methods. Treatment for presumptive UTI based upon UA results alone should be started in the PED as culture results will not yet be available. Proper follow-up should be arranged to monitor the efficacy of therapy and modify it, if needed.

## KEY POINTS

- Urinary tract infection is the most common serious bacterial infection in children.
- Accurate diagnosis of UTI requires both urinalysis and urine culture.

- Urine specimens in non–toilet-trained children should be collected by bladder catheterization or SPA.
- A positive UA does not always mean UTI.
- A negative UA in young children does not exclude UTI.

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# **A BUNDLE OF JOY! THE SICK NEONATE**

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**ROBERT VEZZETTI, MD, FAAP, FACEP**

There is not much in emergency medicine or pediatrics that frightens a clinician like a sick neonate (an infant <28 days old). The differential diagnosis is very broad, and many times, the actual diagnosis will not be evident in the emergency department (ED). The important thing to remember is to have a systematic approach to evaluation and keep calm while treating the patient in front of you.

The most common concern when confronted with a sick neonate in the ED is sepsis. While a temperature of 100.4°F (38°C) or more automatically triggers the traditional septic workup in neonates, there are times when the signs of sepsis may be subtle, such as decrease in feeding or activity. Sometimes parents will report that their child “is not acting right.” If the parents are worried, you should be as well. More concerning is the presence of hypothermia (35°C or less), which can be an ominous finding.

When evaluating a potentially septic neonate, history is extremely important. Be sure to inquire about maternal group B streptococci status in the mother, and also ask if maternal antibiotic treatment was initiated during labor. It is critical to specifically ask about the mother’s herpes (HSV) status, although a large percentage of women do not know their HSV status, so maintain a high index of suspicion.

In neonates that are ill appearing or unstable, defer the lumbar puncture; performing this procedure can cause clinical deterioration. Obtain what cultures you can (typically blood and urine), and give antibiotics immediately. Antibiotic choices in most neonates are ampicillin, gentamicin, and acyclovir (if any suspicion of HSV). Some clinicians use cefotaxime to

replace gentamicin as gentamicin does not penetrate the cerebrospinal fluid (CSF) well. Whether or not to routinely start acyclovir is a matter of some debate. A general guideline is that if there is any suspicion of HSV or if the child is particularly ill appearing (certainly if there are seizures), start acyclovir immediately.

There are multiple metabolic syndromes that can present in the neonatal period. Like sepsis, metabolic disease can be subtle with just decreased activity or feeding. In these neonates, obtaining a venous blood gas, ammonia, thyroid function, and electrolytes (including liver function tests) is important. Many of these infants are hypoglycemic; treat with 5 cc/kg of D10. It is very useful (and courteous) to obtain several red and gold top tubes for future use by the inpatient team when drawing labs. That being said, don't worry if you can't get the blood—treat the child and worry about the details later.

Beware the “septic” neonate who has a murmur (which you may or may not hear), is normothermic, and is around 1 to 2 weeks of age. This is the age for a ductal-dependent cardiac lesion to present, and these neonates are typically hypoxic. Beware that a chest radiograph may or may not show an abnormal cardiac silhouette, so don't let a normal radiograph eliminate the diagnosis from consideration. Strongly consider giving prostaglandin E2 to keep the ductus open and allow for rapid improvement. When giving prostaglandin, be aware that side effects may include hyperthermia, apnea, and a lower seizure threshold. If the child will need to be transported, consider early intubation.

Neonatal seizures can often be subtle. If you suspect a seizure, consider meningitis, metabolic causes, hypoglycemia, or trauma (especially nonaccidental trauma). Of particular importance is infection with HSV, which will often present with seizures. A complete metabolic profile may be helpful in seizing infants, revealing hypoglycemia (worrisome for sepsis, metabolic conditions, or improper mixing of formula), hypocalcemia (transient, metabolic, or nutritional issue), as well as liver function tests (which may be abnormal in metabolic disease and HSV infections). Initial imaging typically is a noncontrast head CT and is useful to rule out acute intracranial bleeding.

Vomiting is often seen in the neonatal period and is usually due to gastroesophageal reflux disease or overfeeding if the child looks well. Malrotation and volvulus will present with bilious emesis and a shocky neonate, and immediate fluid resuscitation is required. An upper gastrointestinal series can confirm the diagnosis, but if the history and exam are concerning, do not delay surgical consultation for an imaging study.

Respiratory illness is common in the neonatal period, and respiratory syncytial virus (RSV) is the most common cause of viral in neonates. Routine RSV testing is not indicated, and a positive RSV in a febrile neonate should not preclude a clinician from performing a full sepsis workup in an ill-appearing neonate. In particularly distressed neonates, a chest x-ray can be helpful, as diaphragmatic hernias and pneumothoracies can present in the neonatal period, in addition to the more common pneumonia.

Injuries from abuse can mimic sepsis or other conditions. If there is an inconsistent history or suspicious examination findings, always consider nonaccidental trauma. The evaluation will vary depending on local protocols, but typically involves a combination of lab and imaging studies.

## KEY POINTS

- Febrile (or hypothermic) neonates require a full sepsis workup, but do not delay antibiotics if you are unable to complete all testing. Consider acyclovir for particularly ill neonates.
- Consider metabolic disease in ill-appearing neonates and obtain additional blood for future testing.
- Ductal-dependent lesions can mimic sepsis; consider using prostaglandin.
- Consider surgical causes of ill-appearing neonates with emesis. Do not delay surgical consultation in ill neonates with suspected volvulus.
- Consider nonaccidental trauma when evaluating sick neonates.

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## BEWARE PEDIATRIC APPENDICITIS

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**JULIA SCHWEIZER, MD**

### INTRODUCTION

Appendicitis in the pediatric population can be a diagnostic challenge. Patients oftentimes present to the emergency department without a flashing neon sign with an arrow pointing to McBurney point saying “It hurts here.” To make things more complicated, symptoms often overlap with other common pediatric illnesses that cause abdominal pain. Because of this, scoring systems have been developed to help sort through the symptoms. The most commonly used scores are the Alvarado and the Samuel/pediatric appendicitis score or PAS.<sup>1-3</sup> Both scoring systems are weighted and take into account right lower quadrant pain, rebound/cough/percussion/heel tapping, leukocytosis ( $>10,000$ ), left shift ( $>75\%$  neutrophils), fever ( $\geq 38^{\circ}\text{C}$ ), anorexia, nausea/vomiting, and migration of pain to right lower quadrant.<sup>1-3</sup> Each scoring system has a total of 10 points. Patients with scores of 1 to 3/4 (depending on which scoring system you are using) are considered negative for appendicitis. Scores of 4/5 to 7/8 are considered intermediate and require more evaluation. Scores of 8/9 to 10 are considered diagnostic for appendicitis and don’t necessarily require further workup. The initial workup in the emergency department for a child with suspected appendicitis should include a 20 mL/kg NS bolus, complete blood count with differential, metabolic panel, and urinalysis. If your patient’s pain/symptoms completely resolve during the time it takes to complete the bolus and get your labs results, consider your evaluation complete.

### IMAGING

“I’m busy; can’t I just get a CT abdomen/pelvis of all the children with

abdominal pain and vomiting in my waiting room?” The short answer is “no.” Multiple studies address the concern that young children (particularly females) are at a significant higher risk for lifetime solid organ tumor following the ionizing radiation associated with CT scans of the abdomen/pelvis.<sup>4,5</sup> As a result of this concern, ultrasound has become the first choice for imaging an inflamed appendix.<sup>6,7</sup>

Your next question may be “What if I don’t have the ability to obtain a diagnostic ultrasound?” The follow-up question would be “If the patient does have appendicitis, is there a surgeon at your facility who will perform surgery on pediatric patients?” If the answer to this question is no, then consider transferring your patient to a center where pediatric care is offered; they are more likely to have pediatric ultrasound capabilities and experience. If the answer is yes, and the patient’s PAS is >8, consider discussing the patient with your surgeon without imaging. A surgeon’s physical exam in combination with the high score may be all of the evidence required to take the patient to the operating room.<sup>1–3</sup> If the patient’s score is between 4 and 8 and ultrasound is not an option, the surgeon may request a CT scan at this point to diagnose acute appendicitis.

## TREATMENT

Acute appendicitis is confirmed by either high score or definitive diagnostic imaging. Patients may be managed with IV fluids, IV antibiotics (such as piperacillin/tazobactam), analgesics (morphine or hydromorphone), antiemetics (ondansetron), and a surgical consult.<sup>7,8</sup>

Stable patients who fall into the intermediate “gray zone” due to either nondiagnostic imaging or inability to image may be treated with IV fluids, an antiemetic, and analgesics (although some feel this should wait until surgical consult is complete). These patients either should be transferred to a tertiary pediatric center or, if already there, will likely be admitted to either a medical or surgical team for observation and serial abdominal exams off of antibiotics.<sup>7,8</sup>

Unstable patients (patients with vital sign abnormalities/evidence of shock) or patients with ruptured appendicitis should be appropriately resuscitated and started on antibiotics (cefoxitin).<sup>8</sup>

## KEY POINTS

- Using a pediatric appendicitis score is a useful tool in diagnosing acute appendicitis in the pediatric population.
- If a patient's symptoms resolve after you've given the patient fluids and are waiting for the lab results, the patient doesn't have appendicitis.
- First-line diagnostic imaging is an abdominal ultrasound.
- You don't need to have a definitive diagnosis to transfer a patient to a tertiary pediatric center.
- There is no need to give antibiotics prior to surgical consult unless the patient is unstable/septic.

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# **DIAGNOSES NOT TO MISS IN THE ACUTELY LIMPING CHILD**

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Children commonly present to the emergency department (ED) with limp or difficulty walking, and the myriad causes range from benign and self-limited conditions to life-threatening diagnoses. The challenge for the ED provider is to distinguish between these possibilities. A high index of suspicion must be maintained for causes such as osteomyelitis or nonaccidental trauma that can be easily missed and yet have severe consequences for the health of the child.

## **INFECTION**

The rapid recognition of septic arthritis is essential to preserve the joint and prevent long-term disability. While it usually affects the knee or hip, other joints may be involved. Children generally appear ill, are febrile, and are reluctant to bear weight or permit range of motion. Axial loading tenderness, an effusion, and a warm and tender joint may be present on physical exam. A complete blood count (CBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and blood cultures can be useful in any limping child in whom infection is a concern. In septic arthritis, however, CRP is the most useful laboratory study. One study showed the probability of septic arthritis was 74% with the presence of both an inability to bear weight and CRP higher than 2.0 mg/dL.<sup>1</sup> Children with neither had <1% chance. Another study showed 100% sensitivity and 87% specificity for an elevated CRP and a temperature greater than 101°F.<sup>2</sup> Ultimately, however, the diagnosis is confirmed via synovial fluid analysis following aspiration. When considering osteomyelitis, ESR and CRP can help exclude or suggest further investigation such as MRI or bone scan. Higher-risk patients are those with

puncture wounds or ulcers or those who are immunocompromised. Abscesses in various locations, discitis, cellulitis, or myositis may also cause limp in children and should be in the differential.

## **INJURY**

An accurate trauma history may be absent in nonverbal children, unwitnessed events, or victims of nonaccidental trauma. One common injury is a toddler's fracture, which is a nondisplaced spiral fracture of the tibia, often from an insignificant mechanism. Most are in the distal tibia and seen in children from the time they begin walking until about 5 to 6 years of age. Of note, they are usually not associated with abuse unlike other spiral fractures of long bones. Initial radiographs may actually be normal. It is important to be familiar with causes of limp that may be suggestive of nonaccidental trauma. Examples of concerning injuries include fractures in various stages of healing, vertebral fractures, metaphyseal-epiphyseal injuries (i.e., corner fractures), bilateral long bone fractures, or transverse fractures. Maintain a high index of suspicion for abusive injury, and pursue a full evaluation as needed.

## **INFLAMMATION**

The most common cause of nontraumatic limp in a child is transient synovitis (TS).<sup>3</sup> This is thought to be a reactive arthritis predominantly affecting the hip. A typical presentation is several days of limping in a well-appearing, afebrile child following a recent viral illness. The leg is typically abducted and in external rotation. The main differential is septic arthritis, and TS is diagnosis of exclusion. It can be helpful to treat a possible TS patient with an NSAID while the evaluation is ongoing as improvement can aid disposition once the child is able to bear weight. It requires only supportive care and will self-resolve.

## **NEOPLASTIC**

A range of tumors may cause a child to limp. The most common malignant tumors are osteosarcoma and Ewing sarcoma, which are found in the distal femur or proximal tibia. Metastatic lesions from leukemias and lymphomas can also cause acute limping. The symptoms may be subtle and are frequently attributed to other injuries, and suspicion should be increased with no trauma history, rest pain, or pain at night. Numerous benign tumors such as osteoid osteomas, osteochondromas, and bone cysts can cause similar



symptoms. Radiographs in malignancy show no clear margins and benign tumors grow slower and typically manifest sclerotic changes.<sup>4</sup>

## STRUCTURAL

Slipped capital femoral epiphysis (SCFE) usually affects children in early adolescence and is most commonly related to obesity. The diagnosis is typically made with AP and frog-leg lateral radiographs that show the classic appearance of ice cream slipping off a cone at the femoral epiphysis. Legg-Calvé-Perthes (LCP) disease is idiopathic avascular necrosis of the femoral head. Plain radiographs in the early stages are frequently normal, so MRI is the best modality if suspicion is high. The presentation can be similar to that of TS but with a different time course. TS will last 1 to 2 weeks and be self-limited whereas LCP can last 18 to 24 months. ED management of this disorder consists of making the patient non-weight bearing and ensuring orthopedic follow-up.

Causes of limp in a child not located in the lower extremities should also be considered when another cause isn't evident. Examples of these include appendicitis and other gastrointestinal ailments, rheumatologic causes, and spinal abnormalities. If something isn't right and the child can't walk, keep pushing until you have a diagnosis. The child who can't walk shouldn't go home.

### KEY POINTS

- Elevated CRP is the most useful laboratory study for septic arthritis. This combined with an inability to bear weight makes the diagnosis even more likely.
- Always ensure you look at all views of radiographs as subtle findings in pediatric injuries can be very easy to miss. Many fractures will also have initially normal radiographs.
- Always consider nonaccidental trauma as a possible cause when the injury doesn't match the mechanism or the developmental age of the child or is well known to occur with abuse.
- Serious diagnoses such as septic arthritis and osteomyelitis can still have normal laboratory values.

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## DON'T DIAGNOSE SEPSIS IN AN ALKALOTIC INFANT

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LISA D. MILLS, MD AND JULIANNE AWREY, MD

Pyloric stenosis is a common disorder of infancy and is present in about 1 in every 250 infants in the United States. Recognition in the emergency department (ED) is critical to successful management and resuscitation of these young patients. The problem, of course, is that many infants present with vomiting, and almost all the parents think the vomiting shoots across the room. Of course, few of those infants will actually have pyloric stenosis. Some basics of epidemiology are helpful in adjusting your index of suspicion. The disease affects males four times as frequently as females. It also tends to run in families; although the inheritance pattern is not well understood, be wary if a sibling or parent had pyloric stenosis. It is seen most commonly in Caucasians and rarely in Asians.<sup>1,2</sup>

Infants typically present between 3 and 6 weeks of age after being born with a normal pylorus that hypertrophies over several weeks. Generally, they will have an unremarkable medical history prior to their presenting symptoms. The vomiting is initially nonbilious and may seem to be random. Over the course of a few days, the vomiting becomes more frequent, forceful, and more closely associated with feedings. The classic projectile vomiting may only present several days in to the developing hypertrophy. Important is that there should be an absence of diarrhea and fever. The child feeds vigorously and without difficulty until later on in the disease course when the child becomes lethargic from dehydration. Indeed, one of the historical red flags should be an infant who seems hungry and wants to feed immediately after a vomiting episode.<sup>1,2</sup>

The physical exam varies with the timing of the process. Early on, the

infant is well appearing with a good appetite. Later in the course as feeding becomes increasingly difficult, signs and symptoms of dehydration develop. In prolonged cases, patients can develop marasmus, characterized by loss of muscle and subcutaneous mass in the buttocks and upper extremities—for all intensive purposes, they look like a dried prune. Abdominal peristalsis in response to contractions against the closed pylorus may also be observed late in the disease.<sup>1,2</sup>

Patients often present multiple times to various providers over the course of the disease. This doesn't necessarily mean a missed diagnosis, but rather represents the natural history of pyloric stenosis as the patient progresses from well appearing to more ill with increasing pyloric hypertrophy. Common initial diagnoses are normal feeding, formula intolerance, and gastroesophageal reflux. Of note is that ultrasound may not initially show a hypertrophic pylorus, so do not be dissuaded from the diagnosis if a recent visit had a normal ultrasound. Do not delay diagnosis of disease until the presence of the late stages of malnutrition.<sup>1,2</sup>

Classically, but not commonly, physical exam will reveal a palpable “olive” in the epigastric region. With modern diagnostic modalities, the olive is now usually only observed when the patient is under general anesthesia in the operating room<sup>1,2</sup> or after a nasogastric tube is placed and the stomach is decompressed.

Lab investigations will show a hypochloremic, hypokalemic metabolic alkalosis from the loss of stomach acid in a sufficiently advanced case of pyloric stenosis. The alkalosis differentiates the ill-appearing pyloric stenosis patient from a septic patient, who should be acidotic. The alkalosis should prompt the ED provider to consider pyloric stenosis in an ill-appearing infant who presents with vomiting.<sup>1,2</sup>

Ultrasound imaging is currently the standard to make the diagnosis of pyloric stenosis, and the sensitivity and specificity approach 100%. Diagnostic criteria includes a pyloric channel length > 15 mm and pyloric wall thickness  $\geq$  4 mm. Patients are administered a bottle to drink during this study, which facilitates identification of the pylorus. It also allows for a dynamic assessment of function of the pylorus.<sup>3,4</sup>

The definitive treatment for pyloric stenosis is a surgical pyloromyotomy, but this is not an emergent procedure. Instead, the treatment of pyloric stenosis begins in the ED with appropriate resuscitation and electrolyte correction. Intensive care admission may be required due to severity of dehydration and electrolyte disorder, and these abnormalities must be corrective prior to any anesthesia use. Coordination with pediatric

surgeons and inpatient teams regarding proper resuscitation and timing of surgical intervention is a key part of the ED management once the infant is diagnosed.<sup>1,2</sup>

The proper diagnosis of pyloric stenosis requires the ED clinician to take several key actions. The first is to avoid mimics such as reflux in a vomiting child with progressive signs and symptoms. The patient should be critically and independently evaluated before agreeing with a prior diagnosis of feeding intolerance or reflux. Another key action is to inquire about the presence of diarrhea. Patients with pyloric stenosis have no diarrhea, and this presentation should not be confused with acute gastroenteritis. Another pitfall is to presume sepsis in an ill-appearing infant with vomiting—use the acid–base status to lead to the appropriate diagnosis. A history of increasingly projectile emesis, the classic hypochloremic, hypokalemic metabolic alkalosis, and the age of the patient all support a diagnosis of pyloric stenosis and should prompt the diagnosis with ultrasound.<sup>1,2</sup>

## KEY POINTS

- Infants with pyloric stenosis may be well appearing and eat vigorously if early in the course of the disease.
- If alkalosis is present, think pyloric stenosis as opposed to sepsis.
- Absence of diarrhea is a red flag to consider pyloric stenosis as a diagnosis.
- Fluid resuscitation is key to the initial management of pyloric stenosis.
- Don't mistake GERD for pyloric stenosis in the young infant with a suspicious presentation.

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## **BRUE: THE DIAGNOSIS FORMERLY KNOWN AS ALTE**

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**JULIA N. MAGANA, MD AND TAYLOR STAYTON, MD**

One of the most frightening events parents can encounter is their baby choking, changing colors, or appearing apneic. Naturally, it drives families to your emergency department (ED). An apparent life-threatening event (ALTE) was defined in 1986 in very broad terms. Because most appear well and only have a worrisome history in the ED, a 2016 AAP Clinical Practice Guideline (CPG) proposed a more precise term: BRUE (Brief Resolved Unexplained Event). BRUE is defined as an event in an infant <1 year of age when the observer reports a sudden, brief, and now resolved episode with one or more of the following: cyanosis or pallor; absent, decreased, or irregular breathing; marked change in tone; or altered level of responsiveness. Clinicians should diagnose a BRUE only when there is no explanation after conducting an appropriate history and physical examination.

Uncertainty drives many providers to uniformly order tests and hospitalize. Even with shotgun evaluations, the etiology of the event is found in only half of patients and doesn't improve patient-oriented outcomes or prevent future events. A thorough history and physical should drive evaluation. The AAP CPG stratifies higher-risk infants as infants <2 months of age, those with more than one event, events lasting >1 minute, need for CPR, and prematurity (<32 weeks gestational age until 45 weeks postconceptional age). Lower-risk patients are >60 days of age, no prematurity, first BRUE, <1 minute, no CPR by trained medical provider, and no concerning historical/physical exam features. ED observation or admission can be considered in the higher-risk patients with focused testing.

The dilemma is in the well-appearing child with only a historical event—

of course, the ill-appearing child gets the thorough evaluation, treatment, and admission. But no lab or imaging test reliably helps with risk stratification. For lower-risk patients, the only lab the AAP CPG 2016 suggests the provider “may” consider is pertussis testing. A CBC rarely contributes to the diagnosis. Anemia is more common in patients with recurrent episodes, but no study reported a causal role or evaluated the effect of lab results on treatment. A toxicology screen by gas chromatography/mass spectroscopy found medications like over the counter cold medications in 8.4% of patients that were not historically disclosed. Despite this, the role in mass screening and outcomes is unclear. Testing electrolytes, venous blood gas or other metabolic disorder screening without a suggestive history just isn’t helpful. Routine ECG finds an abnormality in only 1.4% of patients that resulted in treatment and “may” be considered. All of these tests add to costs, length of stay, and often patient discomfort.

Gastroesophageal reflux is a frequent cause of choking episodes in infants. Reflux associated with choking often occurs with overfeeding, and a careful feeding history often provides this clue. Consider monitoring the baby in the ED during a feeding, addressing parental concerns, and ensuring pediatrician follow-up. Reflux is a clinical diagnosis and most do not need further evaluation or admission.

If the history suggests a seizure, the clinician should consider a head computed tomography (CT), electrolytes, child abuse evaluation, a neurology consult for possible admission, and/or EEG. Consider an ECG, CXR, cardiology consult, and admission in those with a history of color change, sweating with feeds, respiratory distress, failure to thrive, hypoxia, murmurs, or other concerns for cardiac diseases. Patients with respiratory tract infections are at higher risk of event recurrence. Admission should be based on age and clinical status. A fever, hypothermia, irritability, or poor perfusion should trigger a serious bacterial infection evaluation in relation to the child’s age (often urine analysis/culture, blood culture, CXR, cerebrospinal fluid profile/culture) and possible admission.

A full skin exam should be done on every patient. A child abuse evaluation should be considered on all patients with cutaneous injuries, no clear alternative diagnosis, recurrent events, historical discrepancies, a family history of unexplained death or event, vomiting, irritability, seizures, or if the caregiver called 911. This can include a social history, head CT, dilated retinal exam, toxicology screen, liver function tests, and a skeletal survey. This serious diagnosis is reported in 0.4% to 11% of well-appearing infants presenting to an ED or admitted to the hospital after an event, and it must be considered in every child.

Caregivers of lower-risk BRUE should be educated on CPR resources, educated about BRUEs, and engage in shared decision making. Patients may benefit from a brief observation period with continuous pulse oximetry and serial observations to help the parents feel comfortable. If you have social concerns or parents have concerns that don't improve with ED observation, don't hesitate to admit for longer observation. Higher-risk babies who appear well may benefit from a symptom focused workup and admission. Separate the high-risk patients from the low-risk patients to avoid the error of unwarranted evaluations and admissions.

## KEY POINTS

- This is by definition a scary event for parents. They all deserve a thorough history and physical to risk stratify.
- There is no “one test fits all” for ALTE/BRUE. Evaluation should be guided by symptoms.
- Always consider child abuse.
- In lower-risk patients, consider pertussis testing and ECG. No further testing is uniformly helpful. Lower-risk BRUE patients are safe to briefly observe and send home with follow-up, parent education, and CPR resources.
- In higher-risk patients consider focused evaluation and admission for observation.

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## “KID ECGs ARE NOT JUST LITTLE ADULT ECGs”

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KRISTA YOUNG, MD

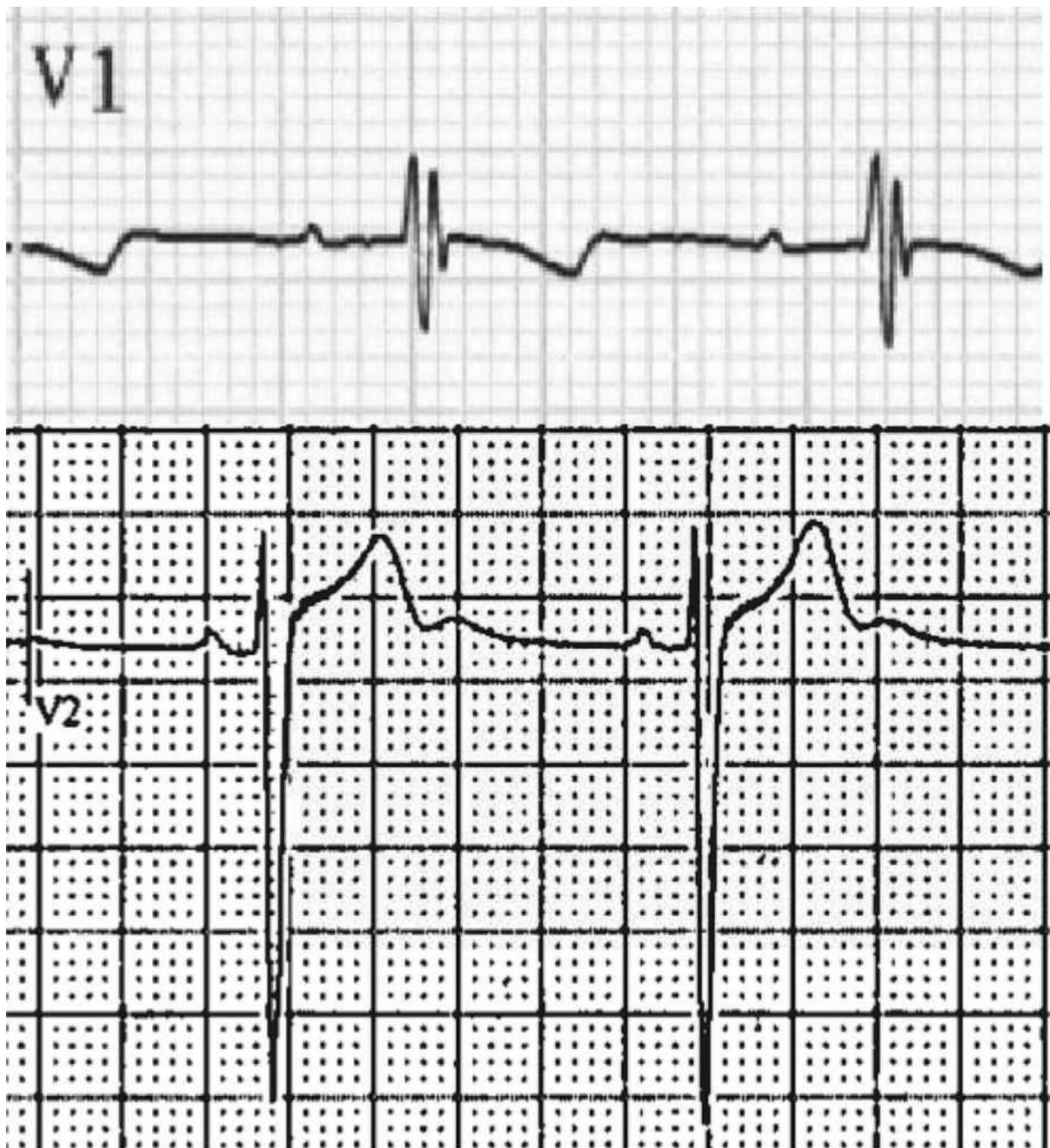
Cardiac physiology changes with age during childhood, and the respective changes can be seen on electrocardiogram (ECG). Some of these expected ECG patterns are only seen in pediatric tracings, and so it is important to differentiate between normal from pathologic. This section will review common findings seen specifically in normal pediatric ECGs.

During fetal circulation, the right ventricle pumps against a relatively high- resistance pulmonary circulation while the lungs are not in use. As a result, the right ventricle is larger and thicker than the left ventricle, and at birth, a neonate’s ECG appears like that of an adult with right ventricular hypertrophy. Specifically, the axis will be rightward; a dominant R wave is seen in V1; Q waves are seen in the inferior and left precordial leads; and the T wave will be inverted in V1-3.<sup>1,2</sup> Each finding will be described further below.

One way to check the axis is to look at leads I and aVF. Normally, the QRS complex will be positive, or up, in both leads. However, in neonates, lead I may be overall negative. After birth, intrathoracic pressures dramatically decrease with the baby’s first breaths, and pulmonary vascular resistance decreases with an increase in systemic vascular resistance. After a few days, the ductus arteriosus normally closes, and systemic output becomes left ventricle dependent. By early childhood or 3 to 4 years of age, the heart takes on the more adult-appearing normal axis with a larger and thicker left ventricle.<sup>1</sup>

Remembering that V1 through V3 are right-sided leads, it is not surprising that a positive dominant R wave is also normally seen in

childhood. Many children also display an rsr' pattern (two peaks/bunny ears), often interpreted as a partial right bundle branch block using adult criteria. In children, if the second peak (r') is not 10 mm greater than the first peak, this is a normal variant that usually disappears by age 5 (See [Figure 324.1](#)).<sup>3,4</sup>



**Figure 324.1** Rsr' seen in V1 with T wave inversion. The slurred ST segment in V2 is an example of J-point elevation.

Likewise, small and narrow isolated Q waves that point downward or are negative can be normally seen in the inferior and left precordial leads (II, III, aVF, V5, V6) in asymptomatic, otherwise healthy patients. However, caution should be practiced, because deep Q waves can be a sign of an underlying pathologic process including myocardial ischemia/infarction, ventricular hypertrophy, and hypertrophic cardiomyopathy. Any patient with new chest pain should be taken seriously especially if several adjacent leads show similar changes and/or if new Q waves are seen that were not on a previous

ECG.

During ventricular repolarization, in kids, the T-wave pattern is correspondingly negative or inverted in V1-3 representing a juvenile T-wave pattern after the first week of life until around 8 years of life. Afterwards, if the juvenile T-wave pattern is still seen into adolescence, this is sometimes a normal variant called persistent juvenile T waves (See [Figure 324.1](#)).<sup>1</sup>

The pediatric heart is also much smaller than an adult heart and must beat faster to maintain cardiac output. Thus, heart rates will appear tachycardic by adult standards routinely above 100 until 4 years of age. Conduction intervals like PR and QRS will also be decreased because less time is needed for the electrical impulse to move through the heart.<sup>1</sup>

Two other special findings that are commonly mistaken for abnormal by those that are not familiar with pediatric ECGs are sinus arrhythmia and benign early repolarization or J-point elevation. When a patient is in sinus rhythm, the ECG shows consistent P-wave morphology in the expected axis for age followed by a QRS. In healthy children, the P-P interval can sometimes vary with respirations and is called sinus arrhythmia (See [Figure 324.1](#)). This finding is physiologically normal and is due to decreases in vagal tone on inspiration causing an increase in heart rate, followed by restoration of vagal tone on expiration. With age, baroreceptor sensitivity and the ability for the carotids to stretch most likely decreases, and the incidence of sinus arrhythmia decreases. Comparatively, in the elderly, sinus arrhythmia is often pathologic due to heart disease or digoxin toxicity and not related to respirations.<sup>3,5</sup>

In J-point elevation, the start of the T wave appears asymmetrically elevated, notched, or slurred after the concordant QRS complex especially in the precordial leads (See [Figure 324.1](#)). In adults, this would be alarming for ST elevation that could represent pericarditis or an acute MI. In healthy children, this represents a benign early repolarization. The physiologic basis is not known, but this finding can be seen until the age of 50.<sup>3,4</sup>

During childhood, specific normal findings are seen on the ECG that represent the healthy growing pediatric heart. Knowing normal from abnormal can be difficult in this population as some of the normal findings mimic concerning findings in the adult population. Remembering normal ECG findings can reassure practitioners, patients, and families alike.

## KEY POINTS

- Due to fetal circulation, in the early stage of childhood, the right ventricle is larger and thicker, and so the ECGs at birth will show right axis deviation.
- Because of the right axis, expect other right-sided findings like juvenile T-wave inversion, R waves in the right precordial leads, or a normal variant of rsr' in V1.
- Isolated inferior and left precordial Q waves can be normal when short and narrow in otherwise healthy children.
- Pediatric hearts are smaller and so have faster heart rates (>100 beats per minute) and shorter conduction intervals.
- Sinus arrhythmia and benign early repolarizations are normal but commonly mistaken during childhood.

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# NOT ALL PEDIATRIC HEAD INJURIES REQUIRE A HEAD CT

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**HEATHER MILLER FLEMING, MD AND KARA KOWALCZYK, MD**

Pediatric head injuries account for nearly 500,000 pediatric emergency visits per year, bringing with them well over a million concerned parents and grandparents who believe their child needs neuroimaging. Indeed, head injuries can lead to significant morbidity and mortality, with those requiring intervention necessitating rapid detection. However, many children who present with the complaint of a head injury do not need imaging, and when grandma asks you, “Why not?” you must be prepared with an answer she will appreciate.

Children are at a higher risk for radiation-induced malignancy due to longer life-span and increased sensitivity of developing organs compared to adults; the younger the child, the higher the risk. Lifetime risk of death due to cancer from one head CT [HCT] is estimated to be between 1:1500 to 1:5000.<sup>1</sup> Given this risk, HCT should only be ordered on patients at risk of clinically important traumatic brain injury (ciTBI). Though definitions vary between studies, most would agree that ciTBI includes one of the following: (1) intracranial injury (e.g. epidural, subdural, and cerebral contusion), (2) neurosurgical intervention, (3) endotracheal intubation for at least 24 hours, (4) hospitalization for at least 48 hours, or (5) death.

In patients with Glasgow Coma Scale (GCS) scores of <14, the risk of ciTBI is as high as 20%. This makes the decision to scan easy; imaging should be obtained in all of these children.<sup>2</sup> But who needs imaging after a minor head injury with a GCS of 15?

Multiple studies over the past 15 years have sought to determine which

patients warrant imaging. The Canadian Assessment of Tomography for Childhood Head Injury trial (CATCH) attempted to address which children of ages 0 to 16 years with minor head trauma required a HCT. The Children's Head Injury Algorithm for the Prediction of Important Clinical Events (CHALICE) sought to derive a clinical decision rule for head injury from a large cohort of pediatric patients in the United Kingdom. These studies demonstrate a high sensitivity for detecting ciTBI, but also have increased CT utilization rates.<sup>4,5</sup> The PECARN (Pediatric Emergency Applied Research Network) rule derivation study is the most robust pediatric clinical decision rule to date, and was a large, multicenter trial that divided preverbal (under 2 years) and verbal (over 2 years) into separate cohorts to determine those patients at low risk for ciTBI who do not need head imaging.

PECARN found that head imaging does **not** need to be performed in children aged 2 years or older with normal mental status, no LOC, no vomiting, no signs of basilar skull fracture, no severe headache and without high-risk mechanism of injury. Conversely, neuroimaging should be obtained in patients  $\geq 2$  years who have altered mental status or signs of basilar skull fracture including palpable skull defect, CSF rhinorrhea or otorrhea, Battle sign, hemotympanum, or raccoon eyes. These children have a 4% incidence of having a ciTBI. Intermediate risk factors for ciTBI in those  $\geq 2$  years old include history of LOC, vomiting, severe mechanism of injury, or headache. Children with intermediate risk factors have a 0.9% incidence of ciTBI and can be observed or HCT can be obtained.<sup>6</sup> Factors influencing this decision are physician experience, multiple versus isolated findings, worsening symptoms, and parental preference. A subsequent validation study found that younger patients, larger hematomas, and nonfrontal hematomas have the highest risk.<sup>7</sup>

HCT should be obtained on all patients  $< 2$  years old with GCS  $\leq 14$  or with palpable skull fracture. All patients under 3 months with any head trauma require imaging due to futility of the neuro exam in this age group. If nonaccidental trauma is suspected, a HCT scan should be performed. Intermediate risk factors for ciTBI in patient  $< 2$  years include nonfrontal hematoma, LOC for  $> 5$  seconds, severe mechanism of injury, or not acting normally per parent. These children can be observed or HCT can be obtained depending on physician experience and parental preference.

Recently, these three decision rules were tested head to head. Though the study population was smaller ( $n = 1009$ ), PECARN proved to be 100% sensitive and 62% specific. Even more interesting was that physician gestalt proved to be markedly sensitive and specific. So, if your gut tells you that something is wrong, go with your gut (and PECARN).<sup>8</sup>

## KEY POINTS

Obtain head CTs on the following kids after minor head injury:

- All kids with a traumatic head injury and GCS are  $<14$ .
- Kids  $<2$  years of age: Palpable skull fracture, hematoma (except frontal), age 3 months or less, or suspected abuse.
- Children  $\geq 2$  years: Altered mental status or signs of basilar skull fracture.
- HCT versus observe: scalp hematoma, severe mechanism of injury, not acting normally, severe headache, vomiting, and loss of consciousness.
- When your gut tells you to get a scan!

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# **EASY DOES IT: BE CAUTIOUS WITH THE CYANOTIC POSTOPERATIVE PEDIATRIC CARDIAC PATIENT**

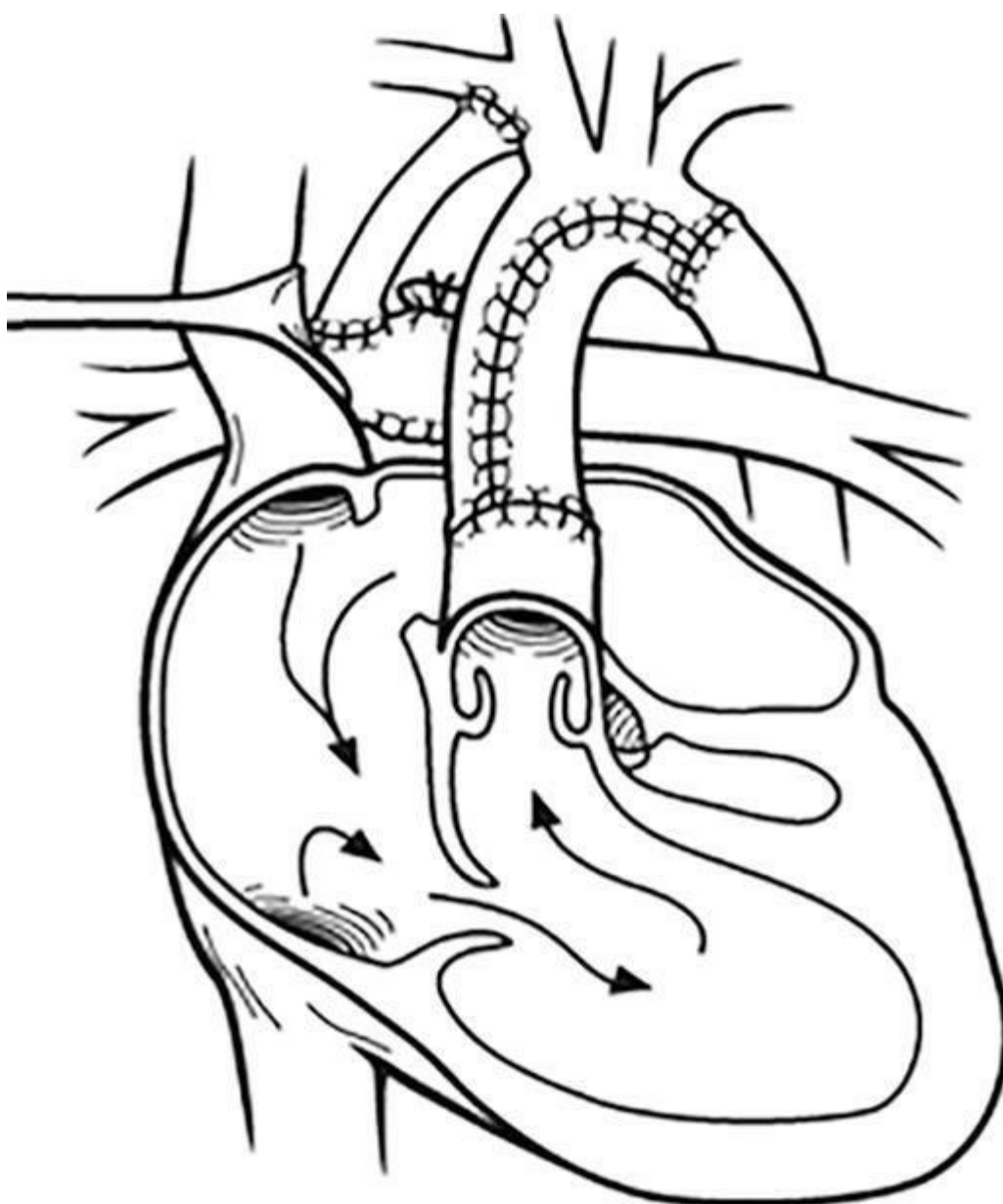
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**TIMOTHY RUTTAN, MD**

Children with complex congenital heart disease are living longer and healthier lives as a result of improved management. One particular subtype that many community ED physicians are unlikely to be familiar with is children with single ventricle physiology. An understanding of the basics of the physiology and surgical repair is critical to avoid catastrophic and potentially life-threatening complications. Case reports exist in the literature of mismanaged children dying in the community ED in part due to a failure to recognize key aspects in the management of these children.

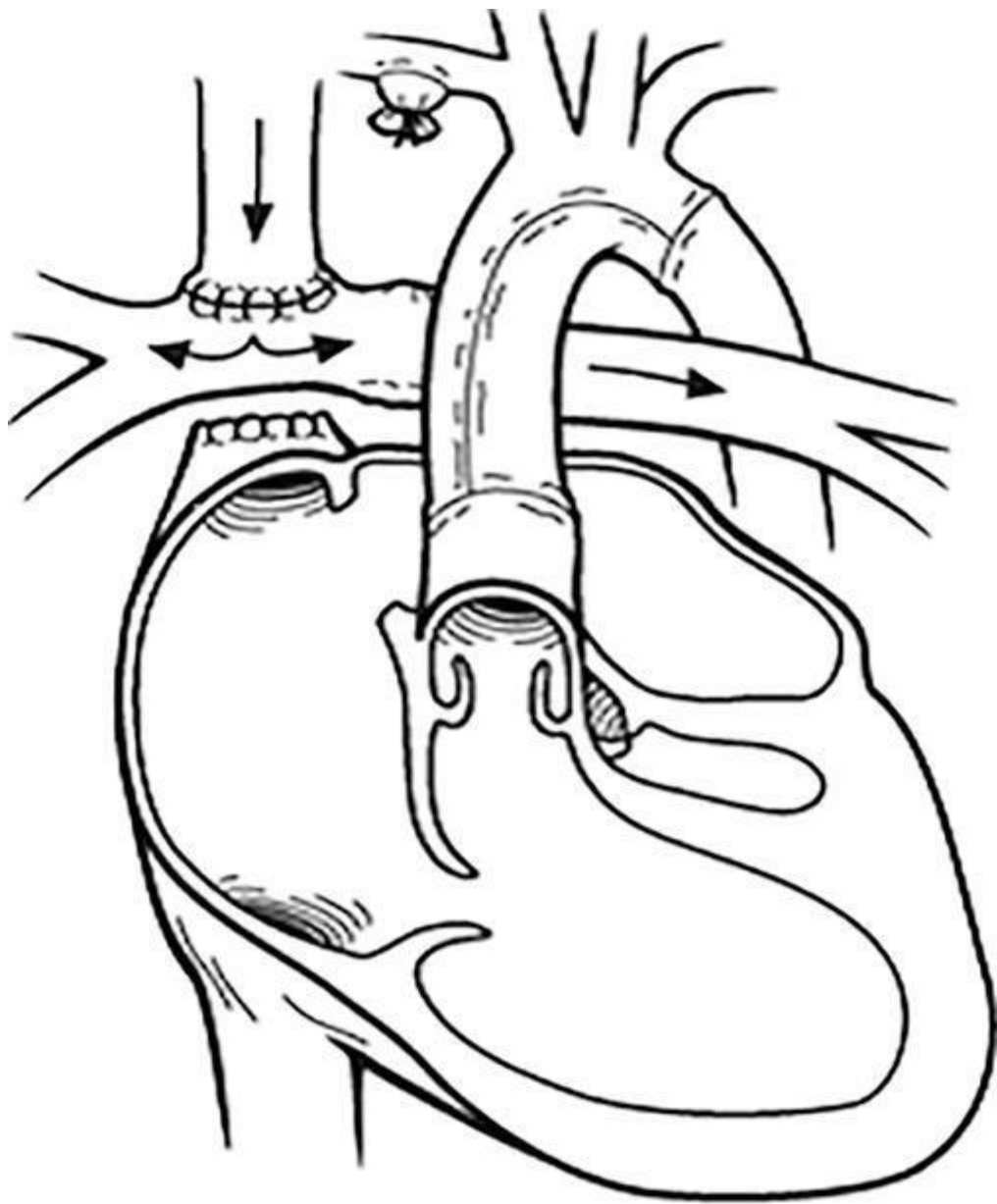
The details of the anatomy and physiology really don't matter for the ED provider, but it is important to have a basic understanding of what happens at the different stages of repair. Hypoplastic left heart syndrome is the prototypical example of single ventricle physiology, although a variety of cardiac lesions have similar repairs. Single ventricle children undergo three surgical stages to allow their one functional ventricle to provide systemic circulation. Pulmonary circulation occurs as a result of passive blood flow driven by systemic venous return. The first stage repair is done prior to the infant leaving the hospital after birth; often referred to as a Norwood (don't remember the names—the details are constantly changing with new surgical techniques), this creates an arterial shunt to provide pulmonary blood flow (see [Figure 326.1](#)). This shunt can be thought of like a permanent ductus

arteriosus, and these children will have a baseline oxygen saturation of about 75% to 85%. This is the most dangerous stage of repair. Once the child makes it to the second stage at about 4 to 6 months of age (often called a Glenn), survival is usually more than 90%. In this repair, the arterial shunt is removed and replaced with a venous shunt that connects the SVC to the pulmonary arteries to give passive blood flow to the lungs (see [Figure 326.1](#)). Since the IVC is not altered, the child has ongoing mixing and remains cyanotic. The third stage of repair usually occurs between 18 months and 3 years of age and is often called a Fontan. This completes the routing of venous return to the lungs where the SVC and IVC are anastomosed to the pulmonary arteries for passive venous return to the lungs (see [Figure 326.1](#)). Once the third stage is reached, the child will have normal or near normal oxygen levels and is not as fragile as the child between the first and second stage.

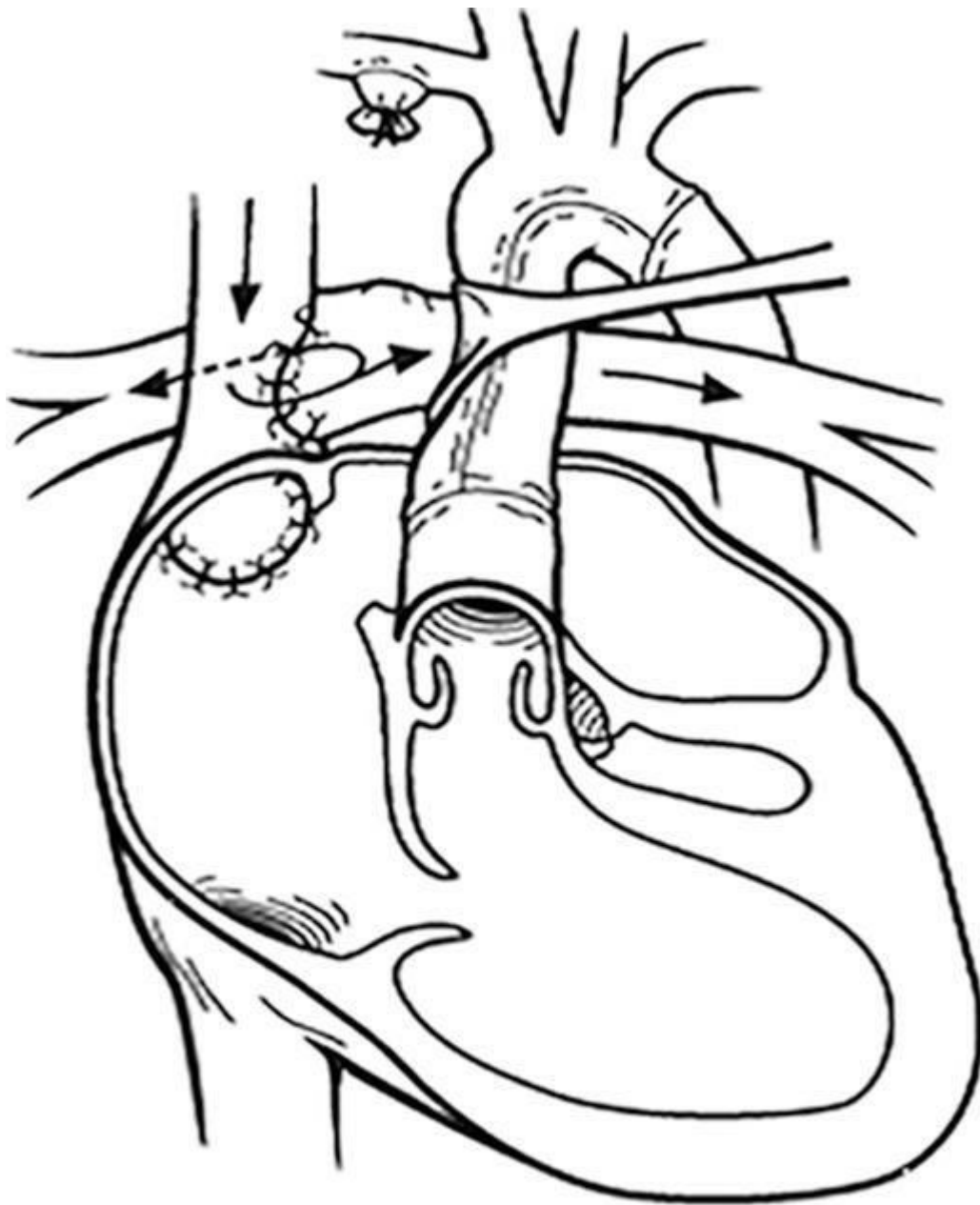


**A**

BT Shunt



**B** Bidirectional Glenn



## C Hemi-Fontan

**Figure 326.1** A: BT shunt. B: Bidirectional Glenn. C: Hemi-Fontan.

That's it for physiology and background. The remainder of this chapter will focus on the child between the first and second stage of repair, as these are the most medically fragile children. The parents should be able to give you two key pieces of information—how many surgeries (stage of repair) as well as the baseline oxygen saturation. With this information and your usual excellent history and physical examination, you can correctly manage these complicated children.

Let the oxygenation saturation guide your understanding of what is going on. In a nutshell, these children are usually either undercirculated (most

common) or overcirculated to the lungs. As can be appreciated from the previous section on anatomy and physiology, blood can go either to the lungs or to the systemic circulation through the shunt in a first-stage repair. If the pulmonary vascular resistance is high or there isn't enough blood (preload), then the lungs are undercirculated. Conversely, if the pulmonary vascular resistance drops, blood floods the lungs and the child can be overcirculated.

The least common problem in the ED is overcirculation of the lungs—unless we cause it. Overcirculation is indicated by an oxygen saturation of more than 85% and means too much blood is getting to the lungs. This can be caused if the shunt is too large or if there are collaterals, but these rarely cause an acute ED presentation. More common is exogenous oxygen that causes pulmonary vasodilation. This is usually our fault; a child comes in with an oxygen saturation of 80% (normal!), and someone slaps a nonrebreather on the child to “fix” the hypoxia. The result is pulmonary vasodilation, overcirculation in the lungs, and a lack of systemic blood flow. If not fixed quickly, this can lead to rapid deterioration and cardiac arrest.

Recall that the single ventricle child is dependent on passive venous return to perfuse the lungs, which makes very prone to dehydration with routine illness. Therefore, the ED provider will more often see undercirculation, which is represented by a pulse oxygenation of less than about 70%. Usually this is from dehydration or a respiratory illness, and less commonly by shunt obstruction or a shunt that is too small. This is where your careful management can save a life. Gently correct any relative hypoxia—usually to 75% to 85%, but don't overshoot or you will get in trouble as noted previously. Most important in the ED, don't be afraid to give gentle fluid boluses. These aren't your adult CHF patients. If these preload dependent children are dehydrated, not enough blood gets to the lungs. Give fluid and reassess—you will be surprised at how it helps. Call the pediatric cardiologist early to help you make decisions on management and disposition, as most often a first-stage repair child will be watched overnight in the hospital to maintain preload.

In short, with single ventricle physiology children, what you do not do matters as much as what you do. Give enough oxygen, but not too much. Give enough fluid, but not too much. Ask the right questions of the parents to help guide your management, and use the pulse oxygen to help you decide if the child is over or undercirculated to guide your management decisions. Most important, don't be afraid of these interesting and complicated children, and you will make a difference in their life.<sup>1-5</sup>

## KEY POINTS

- The parents are your best friend—rely on them for what is normal and abnormal.
- Know the stage of surgery—the name doesn't matter, but be very careful if there has only been one!
- Use the pulse oxygen to guide your management.
- Small changes in oxygen and fluids make big differences.
- Early consultation and consideration of transfer is important—don't be afraid to phone a friend.

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# NOT TOO SWEET: GETTING IT JUST RIGHT IN INITIAL PEDIATRIC DKA MANAGEMENT

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**DARIUSH GARBER, MD, MPH AND NATHAN KUPPERMANN, MD, MPH**

Diabetic ketoacidosis (DKA) is a common reason for children with insulin-dependent diabetes mellitus (IDDM) to present to emergency departments (ED). ED providers must be prepared to provide initial management and resuscitation, while avoiding complications of DKA. Although some complications are similar to those of adults (e.g., electrolyte abnormalities), others are more common in children and are the focus of this chapter. Understanding key differences between children and adults with DKA and risk factors for complications will help the ED provider avoid catastrophe and skillfully manage these challenging young patients.

The most dreaded complication of pediatric DKA is cerebral edema, thought to be secondary to cerebral injury. Clinically apparent cerebral edema occurs in ~0.5% of episodes of DKA in children and is fatal in nearly 25% of affected children. Even those who survive are at high risk for long-term neurologic consequences, making cerebral edema a “worst first” diagnosis to always consider in children with DKA. Children with cerebral edema may present with headaches and vomiting, progressive neurologic changes, cranial nerve deficits, anisocoria, lethargy, obtundation, incontinence, bradycardia, and decreased oxygen saturation. Indeed, some of the earlier of these signs and symptoms are not dissimilar from those exhibited by children who present in DKA without cerebral edema. Once neurological signs develop, however, one must assume that cerebral injury



and cerebral edema have developed.

Cerebral edema has classically been thought to typically occur 4 to 12 hours following initiation of treatment for DKA. However, this complication can manifest as late as 48 hours following initiation of treatment of DKA (despite resolution of ketoacidosis) and has been described in 5% to 10% of affected children on initial presentation to the ED in DKA prior to receiving treatment. Historically, volume and rate of initial fluid administration were thought to play an important role in the development of cerebral edema—emerging evidence over the past decade, however, suggests that this is not the leading cause, and the etiology of cerebral edema in DKA is still not clearly understood. One school of thought suggests that intracellular cerebral swelling occurs due to changes in osmotic gradients with volume repletion. However, recent MRI studies have proposed a more complex etiology where initial hypoperfusion of brain tissue exists in a hypovolemic, low-flow state, with reperfusion injury following fluid administration. This initial hypoperfusion can lead to a phase of extracellular cerebral edema. Ultimately, it is unclear to what degree cerebral edema is based on patient factors versus treatment interventions, but more recent data suggest that the baseline degree of illness of the child plays a very important role. One ongoing large multicenter pediatric trial is seeking to provide clarity on how the rate and content of fluid resuscitation impacts cerebral injury, if at all.

Until there are more definitive answers, we must address the patients in front of us with the available evidence. Established risk factors for DKA cerebral injury and edema based on epidemiological studies show that age younger than 3 years, new-onset diagnosis of IDDM, and severely abnormal laboratory values (e.g., pH < 7.1, low serum bicarbonate, high blood urea nitrogen, low PCO<sub>2</sub>, failure of serum sodium to rise with treatment) are associated with increased risk of developing cerebral injury/edema. Treatment with intravenous bicarbonate is an avoidable risk factor, as well. Essentially, children with DKA who are more ill at presentation are at higher risk for developing cerebral edema, and must be monitored with great vigilance.

When cerebral edema is suspected, treatment must be initiated emergently. This is a clinical—not radiological—diagnosis, and treatment should be initiated before (or often en route to) advanced brain imaging. In addition to elevating the head of the bed to 30 degrees, therapy should be provided to mitigate increased intracranial pressure (ICP). Hyperosmolar therapy reduces blood viscosity and draws water out of brain tissue, resulting in temporary periods of reduced ICP. Options include intravenous mannitol 1g/kg given over 10 minutes or intravenous hypertonic (3%) saline at 5 to 10

mL/kg over about 10 minutes. The effect of these agents, and which is superior, is unclear, however, due to lack of controlled trials.

Although less acutely life-threatening than cerebral edema, thrombotic events are more common in children than adults with DKA and include cerebral vascular thrombosis (CVT) and deep vein thrombosis (DVT). CVT can occur within hours of ED presentation, secondary to dehydration, vascular stasis, and the hypercoagulable state of DKA. In the absence of contraindications, anticoagulation may be appropriate once DKA-related CVT is diagnosed; prophylactic anticoagulation, however, is generally not recommended. The risk of DVT is more substantial if there is a central venous catheter in place. Younger patients are at greater risk due to smaller vessel diameter and, often, more severe disease on presentation. If a central line must be placed, consider avoiding femoral lines and remove the line as soon as possible.

Ultimately, children in DKA are often acutely ill and may decompensate precipitously even before treatment is initiated. Rapid recognition of the severe complications of pediatric DKA, and the directed therapy is essential.

## KEY POINTS

- Recognize that all children with DKA are at some risk for cerebral injury/edema—the sickest and youngest children are at highest risk.
- Cerebral injury/edema is a clinical diagnosis. Monitor the patient closely for neurological decompensation and—if concerned—treat early with hyperosmolar therapy.
- Avoid using intravenous bicarbonate in almost all circumstances (one exception being symptomatic hyperkalemia). It may harm your patient without resolving the underlying acidosis.
- Be aware of the hypercoagulable state of children with DKA, and be cautious of central intravenous line placement locations so as to avoid development of thrombosis.
- Initial IV fluid repletion will decrease serum glucose, without the need for an insulin bolus. After this, start with a regular insulin infusion at 0.1 U/kg/h, which is effective in eliminating ketogenesis while avoiding hypoglycemia.

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## PEDIATRIC CONCUSSION: A LEVELHEADED APPROACH

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**KENDRA GREETHER-JONES, MD AND ERIN OSIECKI, MD**

Pediatric concussion is a common condition seen in the emergency department (ED) and is responsible for up to 200,000 visits annually in the United States. Common causes of pediatric concussions include daily activities, recreational play, or organized sporting events.<sup>1</sup> A concussion can occur from direct trauma, such as striking the head with a soccer ball, or indirect forces such as a body tackle without the head directly striking another object. The number of children and adolescents seeking ED treatment is on the rise as public awareness has increased, making it imperative for ED providers to understand the basics of initial diagnosis and management. Proper early treatment can play an important role in preventing long-term morbidity from this common problem.

Although concussion can be formally defined as “a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces secondary to direct or indirect forces to the head,”<sup>2</sup> this is hardly an explanation that will provide clarity for most parents (or front line ED clinicians). Defining concussion can be as simple as head trauma that results in disruption of the normal functioning of the child’s brain without altering the structure—that is, a head injury without a bleed or fracture. Diagnosis of concussion relies on identifying signs and symptoms that fall into several broad categories including somatic, emotional, behavioral, and sleep disturbances. Loss of consciousness is not required. Symptoms can include headache, nausea, vomiting, dizziness, balance issues, fatigue, difficulty concentrating, poor attention, vision problems, feeling “in a fog,” amnesia, under or oversleeping, and emotional outbursts or moodiness. These are likely already identified by the parent and may be the

driving force behind the visit to the ED. Baseline testing of high school athletes has shown that many of these symptoms may be present in the adolescent population in the absence of concussion, which makes it more challenging for emergency providers, as a single evaluation cannot determine the severity of concussion.<sup>3</sup> Thus, serial evaluations and good follow-up with primary care providers or specialists in pediatric head injury is essential.

It bears repeating that the diagnosis of concussion is purely clinical. Assessment tools can aid diagnosis; however, most are not specific to the ED setting. Laboratory tests do not currently play a role in evaluation of concussion. As the disruption is functional, imaging studies such as CT and MRI will be normal and therefore not diagnostic. When approaching a pediatric patient with head trauma and suspected concussion, it is critical to evaluate for additional “can’t miss” diagnoses. These include skull fractures, intracranial hemorrhage, and c-spine injuries. A large prospective study by Kuppermann et al.<sup>4</sup> created and validated a decision tool to identify pediatric patients at very low risk for clinically significant brain injury, thus reducing unnecessary radiation exposure in pediatric patients. Factors in this decision rule include signs of altered mental status, GCS 14 or lower, signs of basilar skull fracture/palpable skull fracture, LOC, severe mechanism, vomiting, and additionally for those younger than 2 years old, scalp hematoma (excluding frontal). The optimal duration of observation in the ED for these findings has not been adequately studied and, to our knowledge, no guidelines exist.

Once injuries that require further evaluation and admission have been excluded and the patient is deemed appropriate for discharge, return precautions and anticipatory guidance are essential. Physical and cognitive rest are the mainstay of treatment of concussion, but specific evidence-based guidelines are lacking. Nor is there an evidence-based answer to the frequently asked question of “should I wake my child up?” during the night. Most pediatric patients recover within 7 to 10 days, but some may take longer. The patient should be evaluated serially by a knowledgeable primary care provider to determine when it is appropriate to return to school activities in the setting of ongoing symptoms. Symptoms are commonly controlled for the short term with agents such as an antiemetic for nausea and NSAIDs or acetaminophen for headache.<sup>5</sup> Advise parents that these medications must be discontinued before the child can be truly “symptom free” with regard to returning to school or play. It is also advisable to avoid driving while recovering from a concussion.

In the pediatric population, second impact syndrome is a frequent cause for concern. This is the potentially fatal condition that occurs when the patient experiences additional head trauma (often mild) prior to resolution of

previous concussive symptoms. In order to prevent second impact syndrome, pediatric patients with a concussion should not return to play while symptomatic and never on the same day of injury even in the absence of symptoms. The current recommendation is 1 week of symptom free time prior to restarting physical activity. Return to play should occur in a step-wise fashion. An example of general progression for return to play in ascending order includes rest, generic aerobic exercise, sport-specific exercises without contact, controlled sport-specific practice with contact, full sport-specific activity, restricted game activity, and finally full activity. Athletes that compete at higher levels may have a tendency to prioritize physical activity and may need a reminder that they should be symptom free at full-time school activities before progressing through their physical activities.

Athlete or not, encouraging primary care or specialist follow-up to perform serial evaluations and develop appropriate return to activity guidelines is an important part of ED care to help your patients safely return to their normal activities.

## KEY POINTS

- Concussion is a clinical diagnosis.
- Loss of consciousness is not required for the diagnosis of concussion.
- Consider imaging for additional structural diagnoses when appropriate.
- Cognitive and physical rest are the mainstay of initial treatment.
- Patients must be symptom free at rest (without medication) prior to returning to normal activities.

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**SECTION XXI**

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**GERIATRICS**



# **DO NOT UNDERESTIMATE THE POTENTIAL MORBIDITY OF ABDOMINAL PAIN IN OLDER ADULTS**

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**CHRISTINA L. SHENVI, MD, PhD**

Abdominal pain is the most common non-injury-related chief complaint in the emergency department (ED). Older adults with abdominal pain are more likely than younger adults to have a dangerous underlying cause that could require surgery or that could be deadly, and the risk increases with more advanced age.

## **HAVE A HIGH INDEX OF SUSPICION FOR DANGEROUS ETIOLOGIES**

There are many different dangerous causes of abdominal pain in older adults. These should be considered high in the differential diagnosis, and a focused history and physical should be used to decide what further workup is indicated. In each older adult with abdominal pain, consider the following in the differential: aortic dissection, aortic aneurysm rupture, mesenteric ischemia, bowel perforation, cholecystitis, diverticulitis, and bowel obstruction.

About 50% of older adults who present with abdominal pain require admission, and they are admitted for longer periods than younger adults. Patients who are admitted have a 5% to 10% mortality rate, and 20% to 30%

require surgery or an intervention. The two most common indications for surgery in older adults are biliary disease and bowel obstructions. In younger adults, no specific cause of pain is found during their ED visit 36% of the time, whereas in older adults (>64 years old), only 22% do not have an identifiable cause.

## **DO NOT BE FALSELY REASSURED BY VITAL SIGNS, EXAM, AND LABS**

Older adults may have a “falsely negative” exam. For example, older adults with peritonitis are less likely than younger patients to exhibit the classic signs of peritoneal rigidity, fever, and leukocytosis. In fact, 30% of older adults with peritonitis lack a fever and leukocytosis. Patients can also have a “benign” abdominal exam and still have a serious pathology. For example, patients without focal abdominal tenderness but who are complaining of severe pain could also have mesenteric ischemia, with pain out of proportion to their exam.

On the other hand, the workup should also not end when a mild leukocytosis is found on urinalysis. Older adults are more likely than younger patients to have a positive urinalysis without a clinically significant urinary tract infection. Older adults, particularly those who are incontinent, immobile, or in a long-term care environment, often have chronic incidental pyuria and bacteriuria. If the patient has abdominal pain, it should not be attributed to mild abnormalities on a UA without at least considering and exploring other possibilities.

## **UTILIZE APPROPRIATE IMAGING**

Bedside ultrasound by a trained user can help rapidly identify certain high-risk causes of abdominal pain, such as aortic aneurysms, aortic dissection, cholecystitis, cholelithiasis, free fluid, and bowel obstruction. If the ultrasound is positive, it can help facilitate rapid disposition and consultation with the right surgeon. So next time you see an older adult with abdominal pain, try putting the probe on them to look for these entities. However, in many cases, a CT or formal ultrasound will also be needed. In older adults, the risk:benefit ratio for CT scanning is far more in favor of imaging. Older adults are less likely to have negative downstream effects of the radiation such as cancer formation and are more likely to suffer the negative consequences from a missed diagnosis.

## THINK OUTSIDE THE ABDOMEN

Abdominal symptoms can also occur from problems outside the abdomen. For example, patients with a myocardial infarction may have upper abdominal pain or nausea and vomiting. A patient with left upper quadrant or right upper quadrant pain could actually have a lower lobe pneumonia. Patients with vomiting but a nontender abdomen may have an intracranial hemorrhage. Patients with DKA may present with abdominal pain and vomiting but have nothing intrinsically wrong in the abdomen. Think about things outside the abdomen that could cause pain or nausea.

## AVOID ANCHORING

Patients often try to explain away their pain or symptoms and relate it to something they have experienced. For example, they may attribute their pain to indigestion, or a “stomach bug” that other family members have also had. Do not anchor to what they tell you. They may be correct, but you should still go through the exercise of considering the highly morbid conditions that could be present and performing the necessary exam or testing to rule them out.

### KEY POINTS

- Keep the more serious causes of pain higher on the differential diagnosis, such as aortic dissection or rupture, mesenteric ischemia, bowel perforation, cholecystitis, diverticulitis, and bowel obstruction.
- Be aware that older adults with abdominal infections may lack fever and leukocytosis.
- Do not cut short the workup after finding a mildly abnormal urinalysis, as many older adults have incidental pyuria and bacteriuria.
- Have a low threshold for obtaining advanced imaging for older adults with abdominal pain.
- Think outside of the abdomen.
- Do not anchor to what the patient tells you.

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# **THINK ABOUT ACS IN OLDER ADULTS—EVEN WITHOUT CHEST PAIN**

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**CHRISTINA L. SHENVI, MD, PhD**

Acute coronary syndromes (ACS) can be challenging to identify, particularly in certain groups of patients. In older adults, ACS can present without the “typical” symptoms of sudden-onset, crushing substernal chest pain radiating to the left arm and jaw. The symptoms of ACS can be subtle and nonspecific. You should consider ACS in patients with atypical symptoms and at the very least obtain an ECG.

## **ATYPICAL SYMPTOMS ARE COMMON IN OLDER ADULTS**

Chest pain is still the most common chief complaint among patients with ACS. However, atypical symptoms, such as dyspnea, diaphoresis, nausea/vomiting, and syncope, are also frequent presenting concerns. Among adults of all ages with ACS, chest pain is absent in 42% of women and 31% of men. Among adults aged 65 years and older with MI, 47% of women and 41% of men did not have chest pain. Atypical symptoms are more common among women, older adults, and those with diabetes, hypertension, and heart failure. The nature of the symptoms does not, however, alter the incidence of ischemic findings on ECG.

## **IN PATIENTS WITH ATYPICAL SYMPTOMS, THE**

## **DIAGNOSIS IS MORE OFTEN MISSED OR DELAYED**

Patients who have atypical symptoms tend to take longer from symptom onset to seek care. This delay is compounded by the fact that diagnosis of ACS in patients with atypical symptoms often takes longer, with longer time to first ECG. Of patients found to have a ST-segment elevation myocardial infarction (STEMI), those without chest pain who were transported by EMS received a prehospital ECG 72% of the time, compared with 87% of the time for those with chest pain. Performance of a prehospital ECG led to more rapid diagnosis and shorter time to reperfusion. Many EDs also have protocols to obtain an ECG within 10 minutes of arrival for patients with chest pain. Without chest pain, patients who are having ACS would not trigger the automatic ECG order, leaving it up to the clinician to think about ACS and order one. For these reasons, patients who have atypical presentations of ACS tend to have longer door to ECG times and therefore longer time to percutaneous coronary intervention (PCI).

## **PATIENTS WITH ATYPICAL SYMPTOMS HAVE WORSE OUTCOMES**

Even after the diagnosis of ACS is made, patients with atypical symptoms are also undertreated and have worse outcomes. For example, for patients with STEMI, those with atypical symptoms (without chest pain) receive fibrinolysis or PCI 37% of the time, compared with 67% of the time for those with typical presentations. Patients without chest pain were also less likely to receive an aspirin within 24 hours and less likely to receive statins and beta-blockers at hospital discharge. Similar trends hold true for patients with Non-STEMI and unstable angina. Fewer patients who present without chest pain receive PCI, low molecular weight heparin or heparin, aspirin, beta-blockers, or statins.

Given the delays in identification of MIs in patients with atypical symptoms, and their undertreatment, it is not surprising that mortality is also higher in patients who have atypical symptoms. In older women presenting with chest pain and MI, mortality is 13%, but it is 21% for those without chest pain. For men, it is 7% for those with chest pain and 22% for those without it.

Older adults, particularly the very elderly tend to have higher rates of atypical symptoms of ACS but ironically may benefit more from early

invasive therapy for high-risk NSTEMIs. The odds ratio for death or nonfatal MI in adults 75 and over is 0.44 with invasive management over conservative therapy. By contrast in adults age 55 and under, there was no clear advantage of the early invasive management.

## KEY POINTS

- Consider ACS in patients who present with dyspnea, diaphoresis, syncope, or nausea/vomiting, even if they do not have any chest pain.
- Have a low threshold to obtain an ECG in patients with these atypical symptoms, but particularly in those who are older, female, or have underlying diabetes.
- Once ACS is identified, treat patients with atypical symptoms just as aggressively as you would in patients with active chest pain.
- Older adults with high-risk NSTEMI are more likely to have atypical symptoms than younger patients but may benefit more from early invasive therapy.

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# **ACS THE GERIATRIC PATIENT: ATYPICAL IS TYPICAL TREATMENT DIFFERENCES IN ACS IN THE GERIATRIC PATIENT**

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## **DEMOGRAPHICS: WHY WORRY ABOUT ACS IN GERIATRICS**

Emergency medicine (EM) is quickly becoming the specialty of geriatric emergencies. You may not have realized when you began your training and practice in EM that the percentage of geriatric emergency department (ED) presentations and geriatric hospital admissions from the ED are growing all over the world, and geriatric patients, similar to the old adage about pediatric patients, are not just “old adults.” Geriatric patients with ACS present differently, are assessed and treated differently, and are admitted differently than younger patients. Therefore, emergency physicians must increase their knowledge of presentation and treatment of life-threatening conditions in geriatric patients and increase their knowledge of differences in ACS in particular for older patients

## **DIFFERENCES IN PRESENTATION OF GERIATRICS PATIENTS WITH ACS**



Despite the enormous amount of information published about ACS, only a minority of large published clinical trials include geriatric patients: geriatric patients typically account for a disproportionately small number of the study population, and age-subset-specific results are often not reported. Individuals over the age of 75 years account for 6% of the US population but over 60% of deaths from myocardial infarction.

Contrary to the “classic presentation” of the middle-aged ACS patient, ACS is really a disease of geriatric patients. Approximately 33% of all ACS occurs in geriatric patients over 75 years old, and about 80% of all ACS mortality is in geriatric patients; this incidence of geriatric ACS is only projected to increase.

While chest pain is the most common symptom of ACS in all age groups, geriatric ACS patients more often present with atypical symptoms like dyspnea, dizziness, infection, dehydration, and abdominal pain and vomiting; these are further complicated by co-presentation with common comorbidities in elderly patients such as renal insufficiency, diabetes, cerebrovascular disease, heart failure, and dementia.

A high index of suspicion is necessary to diagnose ACS in the elderly, with increased suspicion of atypical presentations and the effects of significant comorbidities in order to reduce delayed diagnosis and worse outcomes.

## **DIFFERENCES IN ECG IN ACS IN GERIATRIC PATIENTS**

The ECG is nondiagnostic in 43% of patients older than 85 years with non-STEMI compared with only 23% of patients younger than 65 years. Left bundle-branch block (LBBB) is present in 34% of patients older than 85 years and with STEMI compared with only 5% of those younger than 65 years making diagnosis harder. Because ECG abnormalities are relatively common at an advanced age, it is particularly important to obtain old ECG results whenever possible so that findings in the ED can be compared with previous changes and interpreted accordingly.

## **DIFFERENCES IN TREATMENT**

Perhaps because of atypical presentations, diagnostic difficulties, ECG changes, and a less clear risk/benefit ratio, age itself is related to lower use of recommended medical and interventional therapies in ACS for geriatric patients, even after controlling for atypical presentations and ECG changes.

Specifically, the likelihood for treatment of ACS in geriatric patients with aspirin and beta-blockers decreases by 15% and 21%, respectively, for every 10 years of increasing age after aged 65 years. This is despite studies that show that benefits of adjunctive therapy (antiplatelet, beta-blockers, ACE inhibitors, and statins) are as great, if not greater, in older adults as in younger adults.

Older adults may gain greater absolute benefits with an early invasive strategy compared with younger adults because of their higher risk for adverse outcomes with conservative management (often despite increased procedural risks). Reperfusion for STEMI provides clear mortality benefit compared with no reperfusion up to age 85 years and possibly beyond. Selection of thrombolytics versus percutaneous coronary intervention (PCI) is based on availability, timing from symptom onset, risks, and benefits, but in general, primary PCI is favored.

Older adults are at higher risk for adverse outcomes including mortality, bleeding, heart failure, and mechanical complications of infarction than younger adults. Older adults have a particularly high risk for death with a 15-fold increase from age 45 to 85 after adjustment for disease severity.

In general, treatment decisions should rely more on a thorough evaluation of comorbidities, functional status, and quality of life.

## KEY POINTS

Geriatric patients with ACS presenting to the ED show significant differences in presentation, diagnosis, treatment, and disposition when compared to younger patients. In general, older patients are more likely:

- To present with atypical symptoms like dyspnea, vomiting, and/or abdominal pain;
- Are more likely to represent with significant comorbidities, which could occlude or complicate diagnosis;
- Are more likely to present with NSTEMI and/or LBBB on ECG;
- Are less likely to receive proper medical and interventional therapy, despite increased likelihood of benefits;
- Are more likely to be misdiagnosed and to receive inadequate therapy.

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# **FOLLOW YOUR ELDERS' FOOTSTEPS, THEY MAY BE ATAXIC**

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**JENNIFER L. PLITT, MD AND MICHELLE RHODES, MD**

Dizziness in the geriatric patient warrants special attention due to its high prevalence and higher conferred morbidity and mortality when compared to younger age groups. A wide variety of potential etiologies often makes it difficult to pinpoint any one specific cause as it is often multifactorial and is considered a geriatric syndrome. Dizziness has a significant impact on health care costs and strongly increases the risk of falls, the leading cause of accidental death in persons aged 65 years and greater. Often, patients present with vague, nonspecific symptoms. The physical exam and imaging often have suboptimal sensitivity or specificity for identifying the cause. This combined with the life-threatening etiologies that can present as dizziness make it a least favorite complaint of many providers.

Older adults have decreased sensory receptors in the semicircular canals, saccule, proprioceptive end organs, and retina. Visual-vestibular reflexes also become impaired with age, and overall health can be affected by neurologic and muscular diseases and overall deconditioning. Dizziness can occur with any mismatch between these senses and their central integration. In the older patient, dizziness is often attributable to medications and polypharmacy and more easily exacerbated by other underlying medical conditions.

The majority of causes of acute dizziness in the older adult are benign and self-limited. Peripheral causes include benign paroxysmal positional vertigo (BPPV), vestibular neuritis, middle ear infections, and Meniere disease. Systemic etiologies include deconditioning, medication side effects,

thyroid disease, urinary and lung infections, electrolyte abnormalities, postural dizziness, overly aggressive control of blood pressure, and hypoglycemia. Acute coronary syndrome (ACS), stroke, intracranial hemorrhage (ICH), mass, and arrhythmia are the most concerning etiologies. These critical diagnoses account for about 4% to 6% of patients presenting for dizziness.

It is important to take a good history when discussing dizziness, as associated symptoms, duration, alleviating, and worsening factors are extremely important in narrowing your workup and eliciting a diagnosis. Keep in mind, however, that try as we might, many patients cannot define their dizziness into light-headedness or vertigo, particularly for older adults. Find out if the dizziness is new or old. Did it start suddenly and severely or more gradually? Are there discrete episodes, as with BPPV, or are symptoms continuous? Ask about head position, tinnitus, and hearing loss, as these suggest a peripheral source. Gait difficulty and focal numbness or weakness should be investigated as a possible stroke. Always obtain a complete medication list as beta-blockers, calcium channel blockers, and diuretics can cause presyncope and postural hypotension. Muscle relaxants and psychotropic medications are notorious for causing disequilibrium. Don't forget that older patients also drink alcohol. Previous history of cardiovascular or cerebrovascular disease, diabetes, congestive heart failure, and valve abnormalities identify patients at higher risk for more serious causes.

The physical exam in dizziness is key. Look for abnormal vitals and orthostatic hypotension. Be sure to include a complete ear and neurologic exam. You must walk the dizzy patient to evaluate for ataxia, wide-based gait, and Romberg's sign. The head impulse, nystagmus, test of skew (HINTS) exam to differentiate peripheral etiology from stroke has been shown to have good sensitivity in high-risk ED patients. A video produced by the author with instructions can be found at <http://novel.utah.edu/Newman-Toker/collection.php>. In addition, consider performing the Dix-Hallpike test to evaluate for BPPV.

For patients without a clear central or peripheral diagnosis after history and physical exam, laboratory studies are indicated to check for electrolyte abnormalities, symptomatic anemia, thyroid disease, acute kidney injury, or alcohol intoxication. Have a low threshold for cardiac enzymes in high-risk patients or those with abnormal ECG as the geriatric patient with ACS can present very subtly.

Head CT is typically low yield, outside concern for mass or intracranial hemorrhage, and can miss the most concerning etiology acute stroke. MRI

should be strongly considered for high-risk patients with focal neurologic deficits or concerning HINTS exam. Keep in mind that MRI can miss early stroke.

Treatment of dizziness is focused on the etiology, which can be a challenge when you have not narrowed it down at the end of the ED visit. When you are lucky enough to find a cause, it should be treated according to etiology. The Epley maneuver is used for BPPV. Treatment for Meniere disease and vestibular neuritis in younger patients often includes benzodiazepines or anticholinergic agents. These can cause a host of side effects, including falls and delirium, and do not have strong evidence for efficacy. Vestibular rehabilitation is likely the safer way to go.

Older dizzy patients with improved symptoms and a treatable or low-risk etiology can be discharged home from the ED. Arrange follow-up in a timely manner and ensure assistance at home. A home health safety evaluation can be considered for those who have fallen or are at high risk for falls.

## KEY POINTS

- Dizziness in the older adult is often vague and multifactorial and is increasingly recognized as a geriatric syndrome.
- A complete history and physical exam will identify high-risk patients.
- Most concerning diagnoses are ACS, stroke, ICH, and infection.
- CT is typically low yield aside from ICH, consider MRI to evaluate for stroke.
- Consider risks and benefits before prescribing meclizine or benzos for peripheral vertigo.

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## HIP AND VERTEBRAL COMPRESSION FRACTURES

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**SANG KEUN “SAM” YI, DO AND PAUL BLACKBURN, DO**

With an aging baby boomer generation experiencing the frailty of age, an increase in their visits to the ED can be expected. Seemingly, minor falls can cause significant injury and morbidity. Two of the more common injuries are hip fractures and vertebral compression fractures (VCF).

Patients with hip fracture may or may not demonstrate significant pain, depending on baseline mental status, head injury, comorbid illness, or distracting injury. Accurate history taking and response to physical examination maneuvers may be problematic for similar reasons. Caution is necessary to avoid overlooking unsuspected injury or illness.

The affected leg may not show “classic” external rotation and shortening. Gently test hip range of motion, taking care not to exacerbate pain or potential fractures. Note neurovascular status. Confirm there are no fractures distal (femur, knee) or proximal (pelvis, lumbar spine) to the hip. Do not empirically apply traction, which may cause or worsen displacement.

Plain films of the hip and pelvis are the usual initial imaging choice. Pelvic fractures can masquerade as hip fractures. Do not order “frog-leg” views if hip fracture or dislocation is suspected. Advanced imaging is necessary for pain or disability if no fracture is noted.

CT of the pelvis has similar sensitivity to MRI and may be the advance imaging modality of choice in the ED. MRI has far greater sensitivity and specificity than plain films regarding fracture detail and surrounding soft tissue but has disadvantages: cost, availability, longer study times, and possible need for sedation.

Treatment begins with recognition of injury and pain management,



occasionally difficult to achieve with narcotics alone. Studies have shown ultrasound-guided femoral nerve blocks, plus narcotics, can reduce the amount of medications given and improve overall pain management. Normally, ambulatory patients are admitted for pain control and medical management, anticipating surgical repair of the hip. Recent literature demonstrates hip fracture patients older than 70 years have improved mobility 4 months following injury when cared for at a comprehensive geriatric facility.

VCFs are common, with an incidence of 1.5 million/year in the United States. They occur in 25% of all postmenopausal women, increasing to 40% prevalence by the age of 80. Most occur within the thoracolumbar region (60% to 75%), a transition zone of rigid thoracic spine adjacent to mobile lumbar vertebrae. Analogous relationships develop when surgically “fixed” or previously sustained VCFs abut “normal” vertebrae. Sustaining one VCF increases the risk of a second 5-fold and two (or more) 12-fold.

The most common etiology of VCFs is osteoporosis followed by trauma, infection, and neoplasm. The flexion-compression mechanism involving osteoporotic bone rarely leads to retropulsion of elements into the spinal canal; therefore, neurologic involvement is infrequent. Atraumatic VCFs in patients under 55 years should raise suspicion of malignancy.

Several imaging modalities and guidelines exist to evaluate VCFs. However, advanced imaging (CT, MRI) should always be used in the presence of suspected neurologic involvement, for patients with more serious underlying conditions or for patients who are candidates for invasive interventions. Decisions regarding repeated imaging are based on development of new, or changing, symptoms.

- 1) Plain radiographs detect loss of vertebral height, alignment disruption, facet dislocation, and increased interpedicular or interspinous distance (>7 mm). Imaging the entire thoracolumbar spine is common to ensure detection of all VCFs. Disadvantages include inability to detect ligamentous injury or detail fracture extent or type and are inferior in assessing other proximate injuries, particularly soft tissue. Plain films assess fracture progression by comparing sequential kyphotic angulation measurements in upright films. Lines extended from the superior vertebral end plate one level above and the inferior end plate one level below the injured segment comprise the degree of kyphotic angulation.
- 2) CT provides far greater detailed information regarding injury assessment or progression, both to the involved vertebrae and surrounding structures. It is excellent for imaging complex fractures

and determining degree and type of vertebral involvement, including occult injuries.

- 3) MRI is the modality of choice for neurologic involvement or suspected ligamentous disruption due to its superiority in imaging soft tissue. Similarly, MRI is preferred in instances of VCFs from infectious or malignant processes. MRI may also be useful in evaluating VCF age; newer injuries are identified by increased signal intensity from water in the vertebral body.
- 4) CT myelography for assessment of cord compression is indicated when MRI is contraindicated, as in patients with pacemakers.

Simple VCFs rarely require more than conservative management: pain control, physical therapy, and bracing as tolerated. Immobility raises the usual concerns: DVT, infection, general deconditioning, and decreasing bone density. Radiotherapy may provide significant pain relief for VCFs from malignancy. Operative management is indicated for failure of conservative therapy (pain), impending or existing neurologic deficit, or extreme spinal deformity. Minimally invasive vertebral augmentation techniques (vertebroplasty, kyphoplasty) are preferred.

## KEY POINTS

- Frailty—beware of more severe injury with seemingly low-impact injuries.
- Anchoring—the best way to miss a diagnosis is to make a diagnosis. When a fracture is found, don't stop looking for other injuries or conditions.
- Imaging modalities—consider advanced imaging (CT or MRI) if negative plain films.
- Treat pain—recognize and adequately treat pain.
- Special units—when available, disposition the patient to a geriatric care unit.

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# **BE SURE TO BUILD A SAFETY NET AROUND THE WEAK GERIATRIC PATIENT YOU SEND HOME**

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**ERIC M. LEFEBVRE, MD**

Generalized weakness is a common presenting complaint in the ED for older adults and can be frustrating to evaluate. The differential is broad, including both life threats and less serious causes. An overall evaluation strategy built around a careful history and physical exam, screening tests to exclude common dangerous disease processes, and thoughtful discharge planning can help give older adults the best shot at both avoiding the harms of overtreatment and untreated disease progression.

The initial history and physical exam should focus on differentiating acute neurologic process such as stroke or intracranial hemorrhage from other causes of weakness and identifying what ancillary testing to pursue. Inquire about acute changes, trauma, new medications, infectious symptoms, cardiac complaints, and reduced oral intake. Collateral information obtained from families, emergency medical services (EMS), or nursing home personnel is often crucial. Pick up the phone and talk to someone who knows the patient's baseline and what happened today; you may save your patient hours of unnecessary diagnostic testing. Pay attention to the medication list. Polypharmacy is very common in the elderly, and about a third of older adults taking five or more medications will experience an adverse drug event each year. Common culprits are benzodiazepines, anticholinergics, vasoactive medications, blood thinners, insulin, and sleeping aids.

A careful neurologic assessment with cranial nerve examination and motor, sensory, cerebellar, reflex, and gait testing helps determine the need

for head CT. On exam, search for signs of volume depletion or overload, pneumonia, and abdominal pathology. Get a rectal temperature. Oral and temporal temperatures can be unreliable in the elderly, and hypothermia is almost always a marker of badness. Expose the patients and examine all of their skin. It's poor form to send home sacral osteomyelitis. If a systematic screening for delirium using a validated tool is not part of your routine assessment for older adults, add it. The Brief Confusion Assessment Method (bCAM) is validated in the ED. If positive, be sure to search for the medical cause of the delirium.

What's that you say? Your exam is normal? They are not delirious. I can send the patient home now, right? Not quite; there are some diseases that cause generalized weakness that can be very hard to pick up on history and physical exam alone. Hyponatremia, renal failure, anemia, myocardial ischemia, and urinary tract infection (UTI) all come to mind. A basic metabolic panel (BMP), hemoglobin, ECG, and a urinalysis (UA) with reflex culture are probably the minimum necessary set of testing. Many providers would advocate checking a troponin regardless of ECG findings given the prevalence of atypical presentations of ACS in the elderly and low sensitivity of ECG alone. Consider thyroid-stimulating hormone (TSH), erythrocyte sedimentation rate (ESR), and calcium testing on a case-by-case basis. The interpretation of a not-so-clean-catch UA in older patients can be challenging. Many older adults have asymptomatic bacteriuria, and if the patient is without signs of systemic illness, altered mental status, has no fever, suprapubic tenderness, dysuria, frequency, or leukocytosis, it may be safer to arrange close follow-up and wait for the urine cultures. Therapy for UTI isn't benign. The resultant *Clostridium difficile*– or fluoroquinolone-associated delirium harms thousands of patients each year.

When the history, physical, and ancillary testing are unrevealing, it's time to do two things: road test the patient and build a safe discharge plan. The goal of the ED evaluation doesn't necessarily need to be the definitive diagnosis of the patient's pathology but rather to exclude dangerous disease and build the patient a safety net large enough to get them to their next encounter with the medical system. Before discharge to the community, the patient should be able to locomote as well as preevaluation baseline (make sure they use their usual assistive devices). Patients who can't are at increased risk of unplanned ED return. Inquire about the patient's support and resources at home. Does he or she have transportation, food, a way to get in touch with his or her doctor, and someone to check on him if things don't go well over the next 24 to 72 hours? You may need to get creative to get patients the care they need, but avoiding the cost and patient safety risk of an observation admission adds real value to the patient's ED stay. Oh and one

last thing: print the labs, copy the ECG, and write three lines in the discharge summary to the clinic doctor explaining what you think is going on. It will make that “PCP follow-up, 1 to 2 days,” much more useful.

## KEY POINTS

- When assessing geriatric weakness, always inquire about acute changes: trauma, new medications, infectious symptoms, cardiac complaints, and reduced oral intake.
- In geriatric weakness, consider a BMP, hemoglobin, ECG, and UA with reflex culture the minimum necessary set of laboratory testing.
- Consider checking a troponin regardless of ECG findings when assessing elderly weakness
- Always inquire about the patient’s support and resources at home prior to considering discharge.

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# GRANDMA IS LOOPY: SPECIAL CONSIDERATIONS FOR ALTERED MENTAL STATUS IN THE OLDER ADULT

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CHRISTINE R. STEHMAN, MD

In the United States, about half of the emergency department (ED) population is elderly (>65 years old). Most shifts include a geriatric patient who either is altered or doesn't seem to be thinking straight. This patient may have dementia, delirium, or both. What can you do about it?

Dementia is slow-onset permanent cognitive decline. Delirium is a potentially reversible breakdown in consciousness and attention with either perceptual or cognitive disturbances. Delirium occurs in times of stress and is common in elderly patients in the ED with approximately 10% meeting delirium criteria. Delirium and dementia are interrelated: dementia is a main delirium risk factor, and many delirious patients have underlying dementia. While both delirium and dementia can cause loss of independence and increased mortality, this chapter focuses on delirium.

While these patients seem complicated, three simple steps make proper care easier by helping to identify the problem without worsening the patient's condition.

**Step 1: Recognize the problem.** Hospitalized delirious patients have higher mortality rates, longer lengths of stay, and increased loss of independence after discharge making early identification and treatment key. However, the EP fails to recognize delirium in up to 83% of delirious patients, leading to delayed or no treatment and increased mortality.

Delirious, particularly in hypoactive delirium, patients often partially compensate and “sneak” by EPs. To catch delirium, EPs must push past their patients’ compensation using a tool to identify abnormal thinking. There are a number of tools available, with the Brief Confusion Assessment Method (bCAM) being preferred for the ED setting.

Delirium identification tools have four interrelated parts. Delirious patients must have an altered (or fluctuating) mental status and symptoms of inattention and either an altered level of consciousness or disorganized thinking. First, EPs should speak to family or caregivers to identify an acute alteration or fluctuation in mental status over the last day. If none exists, no delirium is present. Next, evaluate attention by spelling, counting, stating the months backward, or squeezing the provider’s hand at specific cues. No delirium exists if the patient makes less than two mistakes. More than two mistakes means, the EP should assess level of consciousness with the Richmond Agitation Sedation Scale (RASS). Anything other than an alert and calm patient (score of 0) indicates delirium may be present. Alert and calm patients need an assessment for disorganized thinking. This involves the patient following commands without demonstration or answering simple questions (Does a stone float on water? Are there fish in the sea? etc.). Delirious patients make more than one error.

**Step 2: Identify and treat cause of delirium.** Delirium has four main risk factors: dementia, hypertension, alcohol abuse, and high severity of illness. In addition, delirium can be triggered by many things. Multiple mnemonics exist for these causes with ABCDEF probably being the easiest because it addresses the reversible causes. A: analgesia as untreated acute or chronic pain can lead to delirium. It is important to do a full examination looking for injuries and tenderness, and a history of chronic pain should be noted. B: bladder (urinary retention or infection) and C: constipation, both of which can cause pain and delirium. D: dehydration commonly leads to delirium because of decreased perfusion. Vital signs, skin, mucous membranes, and electrolytes may suggest this diagnosis. E: environmental factors such as noise, heat or cold intolerance, lack of vision or hearing aids, poor sleep, restraints, or inability to get around can upset the fragile mental balance of geriatric patients. Finally, F: pharmacy as many medications can lead to delirium. Applying one mnemonic to any potentially delirious patients can help identify potential causes and guide treatment and further evaluation.

**Step 3: Treat delirium.** ED visits are loud, chaotic, and stressful for even the healthiest patient. For patients who are under physiologic stress and are not able to think normally, the ED can be torture. Delirium treatment involves nonpharmacologic and pharmacologic therapies. Nonpharmacologic



treatment is first line and involves minimizing the chaos of the environment. The Hospital Elder Life Program (HELP) involves four goals and has been shown to prevent and decrease the number of episodes and days of delirium. HELP includes maintaining orientation to surroundings; meeting nutrition, fluid, and sleep needs; promoting mobility; and providing visual and hearing adaptations. Family members can help meet these goals.

Patience is key when caring for delirious patients. Speak slowly face to face while making eye contact. Reorient the patient at the start of the conversation and use short, simple, and, as necessary, repeated explanations. Avoid physical restraints: they increase agitation, increase injury risk, decrease mobility, and prolong delirium.

Hyperactive delirium patients who are agitated and at risk for self-harm or severely interfering with treatment require pharmacologic treatment. First line is the lowest starting dose of a neuroleptic agent (haloperidol 0.5 mg PO/IM, ziprasidone 10 mg IM, olanzapine 5 mg PO). These agents may decrease the severity and duration of delirium. Keep opioid and benzodiazepine use to a minimum.

## KEY POINTS

- Recognize delirium, and if uncertain, treat as delirium.
- Find and treat delirium cause.
- Nonpharmacologic treatment works.
- If agitated, antipsychotics help. Restraints don't.
- Avoid benzodiazepines unless in alcohol withdrawal.

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# **THE GERIATRIC TRAUMA PATIENT IS SICKER THAN YOU REALIZE**

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**REBECCA MILLIGAN, MD AND MICHELLE RHODES, MD**

The fifth leading cause of death in the elderly population is trauma, accounting for 23% of all trauma admissions. When adjusted for injury severity, geriatric patients have a higher level of morbidity and mortality across all severity levels. Falls are the most common mechanism of injury and are the most common cause of unintentional injury and death among the elderly, followed by motor vehicle accidents.

Multiple factors place the geriatric population at a high risk for traumatic events. Chronic illnesses predispose patients to weakness and deconditioning. Impaired vision and gait instability occur frequently, increasing susceptibility to falls. Medications like antihypertensives and psychotropics are associated with trauma. Polypharmacy is linked with increased risk of falls. Many of these patients are on anticoagulants, which can cause injuries to be more severe and make resuscitation more difficult.

Geriatric trauma patients often are undertriaged because the mechanism of injury is seemingly insignificant. Morbidity and mortality are improved when a geriatric patient is taken immediately to a high-level trauma center for care. Geriatric patients may report less pain for the same injury in younger patients that may be falsely reassuring.

Identification of an underlining cause of your patient's trauma is essential. Was the fall with loss of consciousness actually syncope? Was the single vehicle collision caused by an episode of arrhythmia or a seizure? Even seemingly simple falls can be harbingers for future trauma and morbidity. Efforts to prevent future events such as a home health safety evaluation should be strongly considered.

Advanced Trauma Life Support principles apply the treatment of the geriatric patients, though with specific focus on occult injury and lower threshold for trauma center referral and activation. Airway, breathing, circulation, disability, and exposure/environment each have geriatric-specific considerations in the older adult.

Anatomically, the geriatric airway can be difficult to manage. Edentulous patients are difficult to ventilate with a bag-valve mask (BVM). If present, leave dentures in place for BVM. During endotracheal intubation, be prepared for potential for limited mouth opening and decreased neck mobility. Utilizing the video laryngoscope for intubation is often a safer approach in these patients.

Breathing and ventilation require careful attention. Comorbid disease such as chronic obstructive pulmonary disease (COPD) as well as the aging process decrease reserve. Remember preoxygenation, apneic oxygenation, and be prepared for a more precipitous fall in pulse oxygen when intubating. Elders are particularly prone to rib fractures and pulmonary contusions with higher morbidity and mortality resulting from similar injuries sustained by younger adults. Respiratory failure necessitating mechanical ventilation may result in a geriatric patient, whereas a 20-year-old with the same injury may be a candidate for discharge. Strongly consider admission for three or more rib fractures.

The standard hemodynamic parameters are inadequate to determine stability in these patients. Blood pressure increases with age, and the normal parameters for an adult likely represent relative hypotension in the geriatric patient. Increased mortality has been shown among geriatric trauma patients with HR > 90 and systolic BP < 110 mm Hg. Early stages of shock can be masked by the absence of tachycardia secondary to medications such as beta-blockers. Do not defer fluid or blood resuscitation on the basis of unsubstantiated concerns for heart failure and fluid overload. Many older adults are volume depleted due to decreased thirst mechanism and diuretics. Failure to recognize circulatory compromise and aggressively treat increases mortality.

The brain normally loses volume through the aging process, which allows for more brain movement in response to motion, and therefore, more blood may collect in and around the brain before the patient exhibits symptoms. Head CT scan should be used liberally in the geriatric patient. Older patients may have significant intracranial injury despite minor mechanism, normal mental status, and a normal neurologic exam. Both the Canadian and NEXUS II Head CT rules suggest imaging if 65 years or older. Similarly, even falls from low heights can cause severe cervical spine

fractures with rates twice that of younger populations. Be cautious when using C-spine rules to justify no imaging. The NEXUS criteria have been validated in a cohort of geriatric patients with adequate sensitivity; however, the Canadian C-spine rule considers age  $\geq 65$  years a high-risk factor that necessitates imaging studies.

Pay careful attention to environmental exposure and environment. Skin tears and abrasions are caused by less trauma and have greater risk for infection. Older adults are also more prone to hypothermia in a cold open room. Avoid iatrogenic hypothermia with warmed blankets and fluids.

A good secondary survey is key. Orthopedic injuries are common in this population; pelvic and femur fractures have a higher incidence of morbidity and mortality. Radiographs can miss occult fractures, and CT or MRI should be used to evaluate for injuries if the patient continues to have pain despite negative x-rays. As with younger patients, eFAST exam is useful for hemopericardium, hemoperitoneum, and pneumothorax.

Although geriatric trauma patients have higher morbidity and mortality for a given injury, with early recognition of injury, avoidance of undertriage, and aggressive management and therapy, many can return to their preinjury functional status.

## KEY POINTS

- Avoid undertriage
- Identify and treat the underlining cause of traumatic event
- Image liberally
- Do not discharge elders with three or more rib fractures

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# A NORMAL PHYSICAL EXAM DOES NOT EXCLUDE INFECTIONS IN THE GERIATRIC PATIENT

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**DANYA KHOUJAH, MBBS, FAAEM**

There are certain clinical features in emergency department patients that immediately make us think “infection”—fever being a main one. A few others are tachypnea, tachycardia, and localizing symptoms—such as rales in patients with pneumonia or abdominal tenderness in a patient with cholecystitis. It’s a detrimental mistake to depend solely on these features in geriatric patients!

Geriatric patients tend to present atypically with infections. Symptoms such as altered mental status, decrease in functional status, failure to thrive, anorexia, vomiting, frequent falls, or “generalized weakness” are the most common presenting complaints in patients with proven infection.

Fever in the elderly is a very specific finding and is caused by infections 90% of the time, with bacterial infection being the dominant cause with viruses causing <5%. A temperature > 37.8°C in the elderly is associated with markers of serious illness over 75% of the time, such as positive blood cultures or death within one month.

The lack of fever does not exclude an infectious cause for the older patient’s presentation. Less than 20% of elderly patients with proven bacteremia report a fever prior to presentation, and up to 30% do not have documented fevers at all in the ED. This is likely due to the lower baseline temperature, in addition to a blunted fever response among older adults. It has been proposed that baseline temperature decreases by 0.15°C per decade, leading experts to suggest redefining fever in the elderly. Several studies

have looked at investigating a lower fever threshold for the geriatric patient; one study by Castle et al. showed that lowering the threshold of “fever” from 38.3°C orally down to 37.2°C increases the sensitivity of detecting infections in the geriatric from 40% to 83% while maintaining a specificity of 89%. Of note, the most accurate way to diagnose a fever is to label it as an increase from the baseline temperature by 1.3°C (2.3°F) if a baseline temperature is available, such as in nursing home patients. In addition, geriatric patients are more likely to become hypothermic, which is an ominous sign as it is a predictor of mortality in the presence of sepsis.

Tachycardia can be blunted in the elderly, either due to medications that they are taking, such as beta-blockers, or simply due to their decreased ability to mount a catecholamine surge in response to stress.

A sensitive clinical exam finding for an infection in the elderly is the presence of tachypnea, especially in pneumonia. However, studies have questioned the reliability of the triage vitals or electronic monitors in calculating an accurate respiratory rate.

When trying to identify the source of an infection, physicians tend to focus their workup around signs or symptoms that point toward a specific organ system. In the elderly, this is neither sensitive nor specific. For example, 20% of patients with pyelonephritis present with respiratory or gastrointestinal (GI) symptoms. Cough is only present in 50% of older patients with pneumonia. Abdominal tenderness is not necessarily present in patients with intra-abdominal pathology. A quarter to a third of elderly patients with cholecystitis, appendicitis, and diverticulitis will present to the emergency department without any abdominal tenderness on exam. Perforation can also occur without pain or fever. In addition, one-third of emergency geriatric patients with bacteremia will not have an identified local source of infection. That being said, a thorough physical exam needs to be done, looking for skin lesions (specifically bed sores), ear involvement (such as otitis externa), and soft tissue complications associated with prosthetic devices.

In conclusion, be vigilant for subtle signs of infection in the elderly, and don’t be fooled by a “normal” exam!

## KEY POINTS

- Have a high clinical suspicion for infections in the elderly as they present atypically.
- Fever is not sensitive for infections in the elderly.



- Redefine fever in the older adult consider an oral temperature in excess of 37.2°C as your cutoff for fever.
- Calculate the respiratory rate yourself to identify elderly patients with tachypnea and therefore a possible infection.
- Lack of abdominal tenderness does not exclude intra-abdominal pathology (or even perforation) in older patients.

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# **RESPECTING THY ELDERS: DEFINING, DETECTING, AND REPORTING ELDER ABUSE**

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**PATRICIA BAYLESS, MD**

Elder abuse is a difficult but important topic as the elder population increases in the United States. According to the 2010 census, 14% of Americans were 65 years of age or older, with the proportion expected to increase as baby boomers age. One of the fastest increasing demographic categories was adults greater than age 85. The majority of these adults also reported disability affecting their activities of daily living. The definition and recognition of elder abuse have been difficult for health care providers and other professionals. According to the National Center on Elder Abuse, only 1 in 14 cases are reported to appropriate authorities due to underrecognition. How can we improve our recognition and understanding of the complexities of elder abuse?

There are several types of abuse identified, including psychological or emotional mistreatment, physical abuse, sexual abuse, neglect or abandonment, financial exploitation, and self-neglect. The signs of neglect may be much more subtle than bruises or fractures. The best single definition for elder abuse has been accepted by the World Health Organization as follows:

“Elder abuse is a single, or repeated act, or lack of appropriate action, occurring within any relationship where there is an expectation of trust which causes harm or distress to an older person.”

Caregivers committing abuse are very likely to be family members or friends. Sometimes, the abuse is a continuation of long-standing

dysfunctional relationships or develops as a response to changes in the family's living situation as a result of patient's increasing frailty and need for assistance. Profiles in cases of elder abuse show that many of these abusive caregivers perceive a higher subjective burden of care required by the elder patient including activities of daily living, financial support, and medical care. Most research surveys are completed by mentally cognitively older adults. It has been much more difficult to assess for adults with dementia, thus underestimating the true incidence of the crime. Patients with dementia, particularly those who are agitated or physically aggressive, are at higher risk for abuse. Patients in nursing homes and other health care settings including hospitals are not immune to elder abuse, though most abuse occurs in the home setting.

Although we as health care providers and our colleagues in social services consider ourselves well versed in the detection of elder abuse, studies have shown otherwise. We are less likely to routinely ask the elder patient about possible abuse and to question caregivers about the use of excessive restriction for a patient with dementia. There are tools to improve our detection of elder abuse. The Elder Abuse Suspicion Index (EASI) is one of the easiest and most straightforward tools. Most ED triage protocols incorporate questions to assess patient safety such as "Have you been hurt, threatened or made to feel afraid?" This might provide the opportunity to request additional screening or social service referral to discover elder abuse much like we do for instances of interpersonal violence. Such an intervention has not been studied.

Federal laws allow states to interpret how elder abuse is reported and managed. Nearly every state has requirements for reporting to law enforcement or an agency such as Adult Protective Services. There are federal reporting requirements for nursing homes and other health care facilities. Most states also include indemnification for reports made in good faith by interested persons, health care providers, or other professionals such as social workers. The more educated we are, the more likely we are to identify and report. Penalties for failure to report also vary from state to state. An online guide to your state's resources including laws, agencies, and statistics for elder abuse is available.

Self-neglect is a common form of elder abuse identified in the ED, particularly among those with isolated social situations. Home health agencies are a resource who can provide in-home evaluations and encourage patient's to accept resources that will assist them in maintaining their well-being. It is also useful to provide behavioral health support and mental health evaluation for capacity and/or competency.

One of the major barriers in the management of elder abuse is recognition and acceptance of the abuse by the cognitively intact adult who may decline intervention. This is markedly different when compared to endangered children who may be removed without their assent, more similar to the dilemmas we see in instances of interpersonal violence. Care recipients may be reluctant to leave a dangerous situation for fear of repercussions at their home or relationships, loss of familiar surroundings, or lack of the financial resource. Health care providers are sometimes reluctant to report because of lack of knowledge about signs of abuse, lack of knowledge about reporting requirements, fear of retaliation by caregiver, avoidance of possible legal proceedings, and sometimes even empathy for the caregiver who has the difficult task of providing care.

Our goal is to provide safety and security for the elder population. It is important to improve our awareness in the ED and educate ourselves so we may guard the health and safety of the most vulnerable of our elders.

## KEY POINTS

- Only 1 in 14 cases are reported to appropriate authorities due to underrecognition.
- Caregivers committing abuse are very likely to be family members or friends.
- One of the major barriers in the management of elder abuse is recognition and acceptance of the abuse by the cognitively intact adult who may decline intervention.
- The Elder Abuse Suspicion Index (EASI) is one of the easiest and most straightforward tools to screen for elderly patients being abused.

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# **HOW TO AVOID SNOWING SENIORS: PAIN MEDICATIONS AND PROCEDURAL SEDATION IN OLDER ADULTS**

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**LISA C. GOLDBERG, MD AND MICHELLE RHODES, MD**

Older adults commonly visit the ED for acute pain. In many EDs, older adults are less likely to receive appropriate analgesic medications when compared to younger adults. This is at least partially explained by fear of increased risk of adverse effects to pain medication seen among older adults. Additional risk is derived from age-related changes in body habitus affecting drug distribution, polypharmacy with drug-drug interaction, age-related decline of P450 enzyme, and changes in renal and hepatic clearance. It is a delicate balance between failing to control our elderly patients' pain and snowing them completely.

The goal of pain management in the geriatric patient is to assess the severity of pain, select patient-specific analgesia, and effectively reduce pain. Documentation of pain scores improves outcomes, with the Verbal Numeric Rating Scale being the most commonly administered. Pain should be assessed within 1 hour of arrival in ED, before and after treatment, reassessed if the patient remains in ED > 6 hours, and a final reassessment prior to discharge. It is important to assess for pain in cognitively impaired older adults as well. There are several scores that can be used, but more simply, one can look for painful facial expressions, grimacing, withdrawing, or moaning. Family and caretakers are often aware of signs of pain and can be asked as well.

The American Geriatric Society (AGS) and World Health Organization (WHO) recommend the use of nonopioid medications for mild pain (0 to 3/10). Acetaminophen has a good safety profile but is limited to 4 g per day and not an appropriate choice for patients with hepatic issues. While effective, NSAIDs are problematic in older adults with renal insufficiency, gastropathy, heart failure, heart disease, or risk factors for heart disease. In addition, NSAIDs have multiple important medication interactions. If NSAIDs are to be used, older adults should be screened for contraindications, and dosing should be done at the lowest effective dose and for the shortest period possible.

For moderate to severe pain and in the absence of contraindications, opioid medications are the way to go. A key saying in geriatrics “start low and go slow” applies particularly well here. Starting with doses 25% to 50% lower than the typical adult dose with frequent reassessments will keep your older patients safe.

Oral opioids are typically used for moderate pain (4 to 6/10). Hydrocodone is a good option, though limited in dose by its combination with NSAIDs or acetaminophen in the United States. Oral morphine can also be used, though with caution in renal insufficiency. If you are sending your patient home with oral opioids, add a bowel regimen to prevent a return trip for constipation and even disimpaction.

For severe pain (7 to 10/10) or those unable to tolerate oral medications, parenteral opioids are required. Hydromorphone and morphine are great choices but should be used with caution in the setting of liver dysfunction. Fentanyl may also be used, but its short half-life requires frequent redosing. Opioid medications to avoid in older geriatric patients include meperidine and codeine. Codeine has variable metabolism making effect unpredictable, and meperidine has cardio and neurotoxic metabolites.

Regional anesthesia in the form of nerve blocks using longer-acting sodium channel blockade is a great option for the geriatric patient. Nerve blocks may be used for fracture or dislocation reduction, abscess drainage, and pain management pending surgical fixation. For hip and femoral neck fractures in particular, they reduce need for parenteral opioids and have been shown to be safe and effectively performed in the ED for geriatric patients—both with and without ultrasound guidance.

Ultrasound guidance for femoral nerve blocks. Ultrasound improves accuracy and reduces amount of local anesthetic needed. Emergency physicians can easily gain proficiency in this procedure.

Procedural sedation in the geriatric patients requires caution for reasons similar to those for pain management but can be done safely. The medication

of choice should be based upon the procedure and patient's comorbidities. Propofol is often selected given its ease of titration with rapid effect and recovery. Older adults require about half the usual dose. Ketamine has been used with success in geriatric patients. Be sure to consider if the potential increase in blood pressure and heart rate would be detrimental to your patient. Etomidate can also be used safely with rapid recovery as long as you don't mind the myoclonus during your procedure. For many ED docs, midazolam, typically in combination with fentanyl, is the only option for sedation; however, the longer recovery time and risk of delirium is less than ideal for older adults.

## KEY POINTS

- Assess and reevaluate pain with each intervention
- Start low and go slow with medication dosing
- Consider regional anesthesia
- Consider individual patient factors when selecting sedation drugs

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# THE CONSEQUENCES OF GRANDPA'S LOADED MEDICINE CABINET

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**RYAN GALLAGHER, MD AND STEPHEN THORNTON, MD**

Consider for a moment a typical geriatric patient: he takes aspirin and clopidogrel for his stented coronaries; metformin and multiple insulins for his difficult to control diabetes; some combination of amlodipine, carvedilol, furosemide, and hydrochlorothiazide for his hypertension (depending on which ones he remembers in a given day); naproxen for his arthritis but sometimes it upsets his stomach so he takes promethazine for the nausea and some of his wife's oxycodone/acetaminophen for the pain; and he is taking some other medications too but the family forgot to toss them in his bag (and they aren't sure where some of them are anyway) before bringing him in because he just doesn't seem right after getting dizzy, falling, and hitting his head.

Complications from polypharmacy are a significant concern for elderly patients in the emergency department (ED). This is due to a combination of inappropriate use of high-risk medications, adverse drug effects, and barriers to taking medications as prescribed. Risk is directly related to the number of antecedent medications and is increased when additional agents are added to their regimen. Estimates suggest that 20% of Medicare beneficiaries have five or more chronic conditions and that half take five or more medications. Many drugs carry inherent risks that are increased in the elderly and even more of a concern when potential interactions with other prescriptions are considered. Elderly patients are also more likely to lack the ability to comply with a medication regimen for a variety of reasons, including lack of social support and high prevalence of dementia.

Whenever possible high-risk medications should be identified and avoided. Addition of any new medication should be carefully considered and done cautiously. It is especially important to be sure that additional medications are not being added to treat adverse effects of preexisting medications, often referred to as the prescription cascade. It is estimated that nearly 100,000 hospitalizations occur annually for adverse drug effects in patients over age 65, predominantly due to unintentional overdose; the most common agents involved are warfarin, insulins, oral antiplatelet agents, and oral hypoglycemic agents.

We must also do our best to avoid giving out high-risk medications ourselves. One retrospective study showed that 16.8% of geriatric patients discharged from the ED are prescribed one or more potentially inappropriate medication(s). General drug classes of concern include opioids, antihistamines, nonsteroidal anti-inflammatories (NSAIDs), antidepressants, antiepileptics, antipsychotics, and antibiotics. Of these, the top offenders included promethazine, ketorolac, propoxyphene, meperidine, and diphenhydramine.

The role of an ED provider in the management of chronic medications is controversial and not well defined. Significant alterations to chronic medication regimens in the ED should be made only in consultation with the primary care physician or pharmacists. When evaluating an elderly patient's medication list, it is important to consider the indication for any preexisting medication. Studies have suggested that as many as 60% of geriatric patients are taking medication with an inappropriate or absent indication. Regardless of whether or not any change is made in the ED, it is important to ensure that patients who are discharged have a plan in place for follow-up with a primary care provider to evaluate their medications.

The inherent risk of polypharmacy is further complicated in geriatric patients by the fact that they are often less able to adhere to complex prescription regimens. This increases the risk of adverse effects due to inappropriate medication use. This is especially concerning for drugs that have a narrow therapeutic index such as warfarin, digoxin, and phenytoin.<sup>4</sup> Thus, it is important to not only consider the adverse effect of any added medication but also consider whether the patient or his or her caregiver will be able to administer the medication appropriately. Patient and caregiver education is the key to ensuring the best chance that a medication regimen will be followed. Consideration must also be given to follow-up, and communication with the primary provider is very important as they will be responsible for any future changes to the medication regimen.

In summary, the geriatric patient is at high risk for complications from

polypharmacy due to the medications they are already prescribed, the adverse effects of commonly used ED medications, and challenges to following prescription instructions. In this at-risk population, the risks and benefits of any additional medication should be carefully considered. Adverse drug effects, medical necessity, and likelihood of appropriate administration and follow-up should all be questioned prior to prescribing any new medication.

## KEY POINTS

- Consider common adverse drug effects before prescribing additional medications in the older adult
- Identify and, when possible, avoid high-risk medications in the geriatric patient
- Discuss new medications with the patient and caregivers to ensure appropriate administration
- Ensure adequate follow-up for any changes made in the ED

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# **COMMUNICATING AND UNDERSTANDING THE ELDER PATIENT**

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**COLLYN T. MURRAY, MD AND KEVIN BIESE, MD, MAT**

There are a tremendous number of older adults seeking care in emergency departments (EDs) (~19.6 million), and the CDC estimates a doubling of the geriatrics population over the next 25 years. Providers need to be able to communicate effectively with older adults to take good care of them. While the hectic nature of the ED can limit patient-physician interactions, approaching older adults with an established communication framework will improve the care you provide.

## **COMMUNICATION    QUALITY    AND    SPECIAL CONSIDERATIONS**

Preparation is the key for successful assessment of older adults. Start the interview when you have a few uninterrupted moments. Though older adults can take longer to interview, spending the extra time up front to discuss their current complaints, previous experiences with the health care system, and goals of care can ultimately help focus your treatment course. If you have time and the patient is stable, review recent medical records as they often contextualize the current ED visit. Ensure that the patient has hearing aids or glasses. Consider supplying reader lenses and pocket talkers for your older ED patients. Consider voice frequency. Lower-pitched voices are easier to hear. Speak clearly with your mouth visible allowing for multiple opportunities for comprehension. This is particularly important before

completing cognitive assessments. Finally, sit down. It has been demonstrated that patients believe that doctors have spent more time with them when the doctor is sitting.

Providers must also remember that not all older adults are the same. There are many vibrant older adults as well as those who demonstrate cognitive decline; getting to know your patient will help identify care needs. Symptom presentation can also differ. Physiologic changes associated with aging, multiple comorbidities, and polypharmacy may lead to deviations from textbook presentations of pathology. A high index of suspicion and a thorough history and physical exam are necessary to identify acute illness.

## **COGNITIVE ASSESSMENT**

Knowing your patient's baseline mental status and performing cognitive assessments of your patients are critical components of every interview of older adults. It is dangerous to assume that delays in cognitive function, limited responsiveness, or confusion are a patient's "normal." If a patient presents with features of altered mental status, further investigation is required. Contact family members or caregivers if the patient presents alone. During questioning, determine the patient's mental capacity, orientation status, and if any acute changes have been noticed recently.

Last known normal and baseline mental status are also important particularly in terms of stroke care and disease course. A number of tools have been designed for fast, accurate assessment of mental status and differentiating delirium (characterized by acute inattention) and dementia (chronic memory loss). We recommend the Brief Confusion Assessment Method (bCAM) for assessing delirium. This clinical tool is an adaptation from the Confusion Assessment Method-ICU scale that assesses for delirium through questioning aimed at identifying altered/fluctuating mental status, altered level of consciousness (agitation), and disorganized thinking as indicators for delirium. While features of dementia are slow to develop, delirium points to an acute medical process requiring further investigation and treatment. To assess for memory loss and possible dementia, we recommend the Mini-Cog, a short assessment involving a three-item word recall and clock draw test. The patient is determined to have symptoms of cognitive impairment if they are unable to recall any of the stated words. If the results are indeterminate (e.g., one or two words recalled), the patient is asked to draw a clock. Errors in the clock draw are scored as indicators of dementia. This measure has been validated across a number of settings and correlates with the results of more in-depth assessments.

# PATIENT ADVOCACY

Beyond diagnostics, one of the most important responsibilities for a clinician is patient advocacy. The geriatric population is at risk of and from frequent interactions with the health care system, limitations in self-care, and financial constraints. Serving as a voice for your patient can improve the quality of each interaction. Involve the patient's family or power of attorney in his or her care. Remember to address these patients directly and work to differentiate their wishes from those of their caregivers' as it is estimated that there is only 60% agreement between parties in cases involving severe physical or cognitive impairment. Discussions should address goals of care including desired workup, interventions, and result notifications. When possible, discuss or prime future discussion of advanced directives, living wills, and Do Not Resuscitate/Do Not Intubate (DNR/DNI) forms beyond the brief cardiopulmonary resuscitation (CPR) discussion that happens in the setting of acute illness. In doing so, unnecessary testing and hospital admissions may be avoided ultimately improving quality of life. Case managers are invaluable assets in knowing available community resources. Providers should further take advantage of transition care teams and advances in technology that allow for close follow-up after ED visits.

The current "graying" of our population is bringing elderly care to the forefront of health care discussion. Though there are several factors that may complicate the assessment of our geriatrics population, keeping in mind the aforementioned considerations and cognitive assessment tools will allow better, appropriate care delivery within and beyond the ED.

## KEY POINTS

- Put in the time up front
- Know your patient's baseline mental status
- Use the bCAM and mini-COG assessments
- Know and advocate for your patient

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# **SECTION XXIII**

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# **WOUND CARE**



# DEEP SUTURES: WHEN, WHY, AND WHY NOT?

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**HOLLYNN LARRABEE, MD AND R. ALISSA MUSSELL, MD**

When closing a wound by primary intention, the goals of wound management include obtaining a functional closure with optimal wound strength while maintaining a low risk for infection and minimizing eventual scar formation. Multiple factors affect the clinician's approach to wound management: the location, length, and depth of the wound; the type of tissue involved; the tension across the tissue; the level of contamination of the wound; and the time elapsed since the injury occurred. Ideal wound closure includes achieving apposition of the wound edges while minimizing tension and avoiding inversion or dead space.

The two techniques employed for primary wound closure in the emergency setting are percutaneous (skin) and dermal (deep) sutures. Percutaneous sutures pass through both the epidermal and dermal layers of the skin; dermal sutures pass through the dermis without ever penetrating the epidermis. Both percutaneous and dermal sutures can be placed in an interrupted or continuous fashion. The structural integrity of the repair is determined entirely by the suture material that passes through dermis or fascia. Sutures should not be placed within adipose tissue, as this layer provides little to no support for wound closure.

Dermal sutures can be used alone or together with percutaneous sutures for wound closure. Dermal sutures, alone, are indicated for closure of a wound that will later be covered by a cast, or in special patient populations. This includes patients who develop keloids, patients who have poor follow-up for suture removal, and patients in whom suture removal will be challenging or traumatic (infants). In addition, dermal sutures are often the only technique available to close lacerations involving macerated or avulsed

tissue, in which percutaneous closure is impossible. Deep sutures are also mandatory to repair tissues such as galea, periosteum, muscle, or fascia. While percutaneous sutures alone can close wounds under low or medium tension, dermal sutures are a useful adjunct for gaping wounds or wounds under high tension. Excessive tension across a wound interrupts capillary blood flow to the wound edge and can delay healing and cause local ischemia and cellular necrosis. Placing an interrupted dermal suture in each quadrant of the wound will allow the wound edge to be brought together in apposition while removing the tension across the epidermal surface. This type of dermal placement will also reinforce the tissue enough to allow for early suture removal, which can improve final cosmetic outcome.

When dermal sutures are indicated, proper placement and technique is critical. To place a dermal suture, first introduce the needle close to the base of the dermis and then pass through the more superficial layers of the dermis. The second bite is performed by introducing the needle into the superficial dermis of the opposite wound edge and exiting at the deep dermis fascial plane. Both suture tails should remain on the same side of the cross-stitch, so that the knot buries properly after tying.

Although all sutures have inherent potential to increase the risk of wound infection, dermal sutures are associated with higher rates of infection than percutaneous sutures. Research has demonstrated that buried absorbable sutures increase the infection rate and degree of inflammation in contaminated wounds, even with adequate irrigation. This effect is even more pronounced with a continuous dermal closure, which not only uses a large quantity of suture material but also creates a tight barrier that facilitates infection spreading between the adipose and deep tissues and involving the entire wound before any infection is clinically apparent. Conversely, dermal sutures have little to no effect on infection rates in clean or noncontaminated lacerations. While there are clear indications and many benefits to using dermal sutures for laceration repair, in contaminated wounds, this has to be balanced against the increased risk of infection. Current literature supports using dermal sutures to close dead space only in noncontaminated or minimally contaminated wounds using as few sutures as possible.

## KEY POINTS

- The primary goal of wound repair is obtaining a functional closure while maintaining a low risk for infection and minimizing scar formation.

- The structural integrity of any wound repair is determined by the tension on the wound and the suture material that passes through the dermis.
- Indications for dermal sutures without percutaneous sutures include wounds that will be covered by a cast, wounds in patients who develop keloids, or patients in whom suture removal will be difficult.
- Dermal sutures reduce tension across the epidermal surface allowing for improved blood flow to the wound edges, early suture removal, and improved cosmesis.
- Dermal sutures are associated with higher rates of infection and should be avoided in contaminated wounds.

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# **PITFALLS IN EMERGENCY DEPARTMENT ABSCESS INCISION AND DRAINAGE**

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**DAVID WEIN, MD AND JESSE DUBEY, DO**

Emergency department (ED) management of cutaneous abscesses has traditionally included a generous skin incision, drainage, cavity manipulation, packing, and close wound follow-up. Recent literature has challenged these time-honored traditions and includes a movement toward less invasive techniques, avoidance of packing, and a more conservative approach to the use of wound cultures and antibiotics.

When considering an abscess for incision and drainage (I&D), keep the differential broad and consider the use of bedside ultrasound to localize the abscess cavity. The differential can range from simple folliculitis to a furuncle, carbuncle, or even a complicated abscess with associated cellulitis. Other diagnostic possibilities include arteriovenous malformations, lipomas, lymph nodes, herniated bowel, myiasis, kerion, herpetic whitlow, sporotrichosis, and cat scratch disease.

There are several pitfalls that should be avoided when caring for a patient with a cutaneous abscess. First, do not rely on needle aspiration as a definitive method to treat an abscess. A recent study demonstrated a low success rate for needle aspiration compared with I&D. It is important to also consider patient risk factors and comorbid conditions. The following conditions are associated with higher rates of complications: perirectal abscesses, anterior or lateral neck masses (from congenital cysts), hand abscesses (excluding paronychias or felons), an abscess adjacent to vital nerves or vessels, abscesses located in the central facial triangle, and breast

abscesses. If any of the these are present, a surgeon should be consulted or prompt follow-up for wound evaluation and definitive care be sought within 48 hours.

Another pitfall is the failure to obtain a wound culture when indicated. Wound cultures are typically not required for a healthy patient in whom there is no plan to prescribe post-I&D antibiotics. It is recommended that a wound culture be obtained in the following settings: a severe local infection, systemic signs, a history of recurrent or multiple abscesses, failure of initial antibiotic therapy, extremes of age, immunocompromised state, or if they are from a region of unknown *Staphylococcus aureus* susceptibility or an area of rapidly changing susceptibility.

Not all patients with an abscess require antibiotic therapy. The Infectious Disease Society of America recommends against routine use of antimicrobial therapy in the young healthy population. If antibiotics are indicated, suggested regimens are outlined in [Table 343.1](#).

**TABLE 343.1 COMMON ORAL ANTIBIOTIC TREATMENT FOR MRSA**

| <b>DRUG (PO MRSA COVERAGE)</b>     | <b>ADULT DOSE</b>  |
|------------------------------------|--|
| Clindamycin                        | 300–450 mg PO t.i.d.–q.i.d.  |
| Trimethoprim-sulfamethoxazole      | 1–2 DS Tabs PO b.i.d.  |
| Doxycycline                        | <45 kg: 4 mg/kg PO b.i.d., >45 kg: 100 mg PO b.i.d.                    |
| Minocycline                        | 200 mg once, then 100 mg PO b.i.d.                                     |
| Linezolid                          | 600 mg PO b.i.d.   |
| Tedizolid                          | 200 mg PO once daily   |
| <b>Drug (IV MRSA Coverage)</b>     | <b>Adult Dose</b>  |
| Vancomycin                         | 15–20 mg/kg/dose Q8–12h (Max 2 g per dose)                             |
| Daptomycin                         | 4 mg/kg QD (skin and soft tissue infection)<br>6 mg/kg QD (Bacteremia) |
| Linezolid                          | 600 mg IV b.i.d.   |
| Ceftaroline                        | 600 mg IV Q12h   |
| Dalbavancin (skin and soft tissue) | 1 g IV on day 1, followed by 500 mg IV on day 8                        |
| Tedizolid (skin and soft tissue)   | 200 mg IV QD   |
| Televancin                         | 10 mg/kg QD  |

Packing wounds remain controversial. Several small studies suggest that there is no difference in healing time, wound recurrence, or wound complications when the abscess is left open. A recent systematic review suggested that packing wounds resulted in delayed wound closure. Patients who may still benefit from packing include those with an abscess >5 cm in diameter, diabetes, immunocompromising conditions, or a pilonidal

abscesses. Multiple studies have been performed examining the difference between primary and secondary closure of cutaneous skin abscesses. Current literature favors a secondary healing process as the treatment of choice for uncomplicated cutaneous abscesses.

Lastly, the “loop drainage” technique has introduced the possibility of having one postoperative visit, painless removal of a drain, and the possibility of only two small scars afterward. One initial “stab incision” is placed at the abscess midpoint or the area where spontaneous drainage has occurred. A hemostat is then utilized to break up loculations, and the abscess is irrigated as typically performed. The distal margin of the abscess is then probed followed by a second “stab incision.” The hemostat is then used to traverse the entire abscess cavity subcutaneously, grasp the silicon vessel loop or ¼ inch Penrose drain, and pull the loop through the abscess cavity. The ends are then tied loosely, and the knot is moved from side to side on a daily basis to allow continued drainage of the abscess until removal after 7 to 10 days.

## KEY POINTS

- Consider patient risk factors, comorbidities, and high-risk locations prior to performing an I&D.
- Wound cultures are not required for simple abscesses in the young and healthy.
- Antibiotics are not recommended unless cellulitis or other high-risk features are present.
- Packing the abscess cavity may delay healing and is associated with more pain.
- Consider the “loop drainage” technique, as this requires less follow-up, produces less scarring, and increases patient satisfaction.

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# **KEEP IT CLEAN: PITFALLS IN TRAUMATIC WOUND IRRIGATION**

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**ANAND K. SWAMINATHAN, MD, MPH AND ELICIA SKELTON, MD, MPH**

Proper wound irrigation is crucial for infection prevention, removal of debris, and promotion of proper wound healing. The goal is to remove all contaminants and foreign material, while causing the least amount of damage to the tissue. If irrigation is not done properly, detrimental outcomes to the patient or the provider may result.

## **PERSONAL PROTECTION**

Adequate personal protection is imperative when irrigating any wound. Prior to preparing the patient, the provider should wear a face mask with eye shield and gloves. Disposable gowns may be used based on the estimated risk of provider contamination.

## **PREPROCEDURAL PREPARATION**

Overall success is guided by preprocedural preparation. Appropriate anesthesia is necessary to ensure proper irrigation. Irrigating wounds is an uncomfortable procedure when performed properly. If patients are not properly anesthetized, they may not tolerate high-pressure irrigation. The wound area should be positioned in a way that allows the irrigant to continuously run off, so that the wound does not soak in the irrigating solution, an event that has been associated with increased risk of infection.



## **SKIN PREPARATION**

Cleaning intact skin surrounding the wound may be useful in reducing the amount of nearby bacteria. However, cleaning solutions should never be directly applied to open wounds. Commonly used cleansers such as povidone-iodine, chlorhexidine, and hydrogen peroxide can be toxic to wound tissue. Not only can these cleansers impair wound healing, they may even promote bacterial growth by weakening host defenses.

Wounds that occur in areas of the body that are hairy may be particularly challenging. Hair may obscure foreign bodies and may make it more difficult to explore and repair wounds. Additionally, hair itself may serve as a contaminant. At times, it may be beneficial to trim surrounding hair. Shaving the area with a razor should not be done, as this can increase infection rates.

## **SCRUBBING**

In excessively dirty wounds or in wounds with significant amount of foreign material entrapped, it may be necessary to scrub the wound. If scrubbing is necessary, it is important to avoid overaggressive scrubbing, as this can lead to further tissue damage and poor wound healing. If the decision is made to mechanically scrub, a fine pore sponge should be used to minimize the amount of tissue damage.

## **IRRIGATION**

Irrigation is most important in contaminated wounds and in areas that are more prone to infection, such as areas that are poorly vascularized. Though sterile saline is often used in wound cleansing, tap water irrigation has been shown to be just as effective. Additionally, tap water is cost-effective and may be easily obtained in large quantities (but it is important to note that these studies apply to areas where potable water is available). There is no added benefit to adding a wound cleanser or disinfectant to the irrigant.

## **IRRIGATION TECHNIQUE**

The amount of pressure to use during irrigation depends on both the level of contamination and the wound site. For clean wounds or wounds in areas of loose skin, (i.e., eyelids, testicles) low pressure, 0.5 pounds per square inch (psi), may be used. However, it should be noted that high-pressure irrigation ( $\geq 7$  psi) is more effective for removing bacteria and other foreign contaminants. The most common and cost-effective method for low-pressure

irrigation is the bulb syringe, which typically produces a pressure of 0.5 psi. High-pressure irrigation can be achieved by using a 19-gauge needle or catheter and a 35-mL syringe. High-pressure irrigation, however, is not without risks and has been associated with tissue damage. It is conceivable, although not proven, that under very high pressures, bacteria and foreign matter could spread along the tissue plane and contaminate previously uncontaminated areas. In order to optimize the effects of irrigation while limiting tissue damage, pressures of 5 to 8 psi have been recommended.

Although the optimal amount of irrigation is unknown, it is typically recommended to use at least 200 mL. Another recommended amount is based on length of the laceration and calls for roughly 60 mL per linear centimeter of the wound. More importantly, clinical judgment should be used. It is generally better to overirrigate than to underirrigate. Contaminated wounds and chemical burns will require more copious irrigation.

Finally, time elapsed since the wound occurred should be considered. Waiting for prolonged periods of time to irrigate after the initial injury may result in higher risk of wound infection. One study using an experimentally contaminated animal model demonstrated a statistically significant reduction in bacterial growth with early wound irrigation compared to later wound irrigation. Overall, optimization of wound management should be based on clinical presentation while keeping the key concepts and pitfalls of wound cleansing highlighted in this chapter in mind.

## KEY POINTS

- Preparation and appropriate anesthesia are necessary to ensure proper irrigation.
- Cleaning solutions should never be directly applied to open wounds.
- Avoid aggressive scrubbing, this can lead to further tissue damage and poor wound healing.
- Tap water has been shown to be safe and effective in wound irrigation.
- In order to optimize the cleansing effects of irrigation, copious amount of irrigant and pressures of 5 to 8 psi have been recommended.

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## PLANTAR PUNCTURE WOUND PEARLS AND PITFALLS

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**R. GENTRY WILKERSON, MD**

Plantar puncture wounds are a common presentation to the emergency department (ED). The management of these wounds is controversial due to a lack of a robust body of medical evidence. Much of the current literature is retrospective reviews of patients with wound complications. These wounds often have a benign appearance at presentation and usually have a good clinical course. However, in some cases, these wounds can have devastating long-term outcomes despite appropriate medical care.

Evaluation should begin with a thorough history that includes time of injury, type of penetrating object, type of footwear worn at time of injury, and care rendered by the patient. It is also important to ask if the penetrating object was intact if removed by the patient. An accurate immunization history will determine the need for appropriate tetanus prophylaxis with tetanus toxoid. Patients with no prior, or incomplete, tetanus immunization require tetanus immune globulin.

The physical exam should determine the location of the wound and assess the integrity of the surrounding soft tissue. Neurologic and vascular function proximal and distal to the wound should be evaluated. Plantar puncture wounds can be classified based on location of the foot into three zones: zone 1 extends from the metatarsal necks to the toes, zone 2 extends from the distal calcaneus to the proximal metatarsal neck, and zone 3 overlies the calcaneus. The external surface of the foot should be washed. If the wound is large enough, the wound can be gently irrigated. High-pressure irrigation should be avoided due to risk of causing injury to tissue and potential for pushing retained foreign bodies or bacteria deeper into the wound. Simple probing of the wound may falsely reassure the provider that a

foreign body is absent and also may result in pushing the foreign body deeper into the wound. For simple puncture wounds at risk for contamination or retained fragments, it may be necessary to extend the length of the wound and gently explore and irrigate the area. An appropriate regional nerve block can facilitate wound exploration.

Any concern for retained foreign body mandates further evaluation. The clinician should consider that the retained object might be part of the footwear and not the penetrating object itself. Plain radiographs will detect most metallic objects. Glass fragments larger than 2 mm should be visualized regardless of the lead content of the glass. Ultrasound may have increased sensitivity for detection of wood and other radiolucent objects. False positives can occur with ultrasound as a result of trapped air within the wound, presence of sesamoid bones, and calcifications. Other imaging modalities include computed tomography and magnetic resonance imaging.

Patients who present shortly after injury do not require laboratory studies. Patients who seek medical care in a delayed fashion typically do so because of persistent, or increasing pain or signs of infection. In cases of suspected infection, laboratory tests to consider are a complete blood cell count, erythrocyte sedimentation rate, and C-reactive protein although no laboratory test can rule out infection. In cases of osteomyelitis, a bone biopsy with culture will help direct antibiotic therapy.

The overall risk of infection for plantar puncture wounds is 2% to 10%. The risk of infection increases with increasing depth of wound, zone 1 location, presence of devitalized tissue, presence of retained foreign bodies, delay in presentation >48 hours, and a history of diabetes. Providers must determine the need for antibiotic therapy. The goal of antibiotic prophylaxis is to prevent complications such as cellulitis, abscess, osteochondritis, osteomyelitis, and pyogenic arthritis. The organisms commonly associated with these infections are *Staphylococcus aureus*, beta-hemolytic streptococci, and *Pseudomonas aeruginosa*. Pseudomonal infection is associated with puncture wounds through rubber-soled athletic shoes. There are no prospective, randomized trials assessing the efficacy of prophylactic antibiotics. In one retrospective study of adult patients who developed infectious complications, only half the patients received prophylactic antibiotics. This suggests no benefit to prophylactic antibiotics in an undifferentiated patient population. Patients with diabetes who develop infectious complications have a much worse clinical course than patients without diabetes. In one study, diabetics were found to have a 46-fold increased risk of lower-extremity amputation. Despite the lack of prospective data, diabetics may be a population that benefits from prophylaxis. If the provider opts to begin antibiotics, then methicillin-resistant *S. aureus* and

*Pseudomonas* coverage should be provided.

Patients with simple presentations of plantar puncture wounds can usually be discharged. They should be provided with detailed wound care instructions and given follow-up in 2 to 3 days for wound reevaluation. Some patients may benefit from being given crutches and placed on non-weight-bearing status until the first reevaluation.

## KEY POINTS

- Perform a detailed wound history.
- Extension of the wound may be required to allow for appropriate irrigation and exploration.
- Plain radiographs may locate metallic objects and glass pieces over 2 mm. Ultrasound is superior for detection of other radiolucent objects.
- Uncomplicated, plantar puncture wounds in healthy patients who present early likely do not benefit from antibiotic prophylaxis.
- Diabetics have a greatly increased risk of requiring amputation if an infection develops.

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# DO NOT BELIEVE THE ADAGE THAT EPINEPHRINE CANNOT BE USED FOR DIGITAL BLOCKS

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Finger and toe injuries are common in emergency medicine. These wounds are associated with an increased risk of bleeding, due to the vascularity of the digits. Achieving hemostasis while providing adequate analgesia is essential when treating these injuries. For most finger injuries (i.e., lacerations, nail injuries, tendon repair), a digital block can be performed to achieve analgesia. In addition to these injuries, an ingrown nail, felon, paronychia, subungual hematoma, dislocations, and fractures may require a digital block in order to provide appropriate treatment.

The digital nerve block is an injection of anesthetic at the base of a finger or toe. It can allow for a minimum amount of anesthetic to be used to achieve adequate pain control. In addition, the digital block avoids injection of the anesthetic directly into the wound, which can distort the anatomy, be more painful for the patient, and make repair more difficult. The standard teaching for a digital block has been to use lidocaine, or another local anesthetic, without epinephrine. It has long been believed that epinephrine decreases circulation to the fingertips, leading to necrosis, and possible loss of digits. Therefore, the common adage is to avoid using epinephrine when anesthetizing patients to treat digit injuries.

The original data regarding epinephrine in digital nerve blocks come from the late 19th to mid 20th century. There are multiple case reports of epinephrine use that were associated with digital necrosis. The majority of the studies were not performed in the emergency department, nor were they

designed to determine the safety of epinephrine. Upon further review of these cases, it is likely that digital necrosis occurred due to infection, tourniquets, or older anesthetics such as cocaine and procaine. Furthermore, the amount of epinephrine used was unclear in a majority of these cases. No cases of digital necrosis have been reported with the use of more recent formulations of lidocaine and epinephrine. In fact, a recent literature review, which included 12 randomized control trials, found that epinephrine (1:100,000–200,000) is safe for use in digital nerve blocks in most patients. Furthermore, there have been no reported cases of patients with poor peripheral circulation harmed by epinephrine, although the majority of studies excluded patients with peripheral vascular disease. The author concludes that the risk of vasoconstriction is overstated. There have also been retrospective cohort studies in podiatric patients that had over 250,000 combined epinephrine injections with no reported complications.

As aforementioned, achieving hemostasis is important in the evaluation of digit injuries to allow for thorough exploration of the wound. The vascular supply of the fingers is from digital arteries that run along the ulnar and radial side of each finger. Each digit is innervated by four nerves, which arise from the median or ulnar nerves. The toes have similar innervations that arise from the tibial and peroneal nerves. The benefits of epinephrine include a faster onset of anesthesia, as well as a prolonged analgesic effect. Lidocaine (amide group) is the most commonly used anesthetic for digital blocks. Lidocaine can be combined with epinephrine (lidocaine 1% or 2% with epinephrine 1:100,000 or 1:200,000) for anesthesia. Since the digital arteries run in close proximity with the digital nerves, it is possible to induce vasoconstriction of the arteries, although this effect tends to wear off after 60 to 90 minutes. Longer-acting anesthetics, such as bupivacaine, can also be used if a prolonged duration of action is needed (4 to 8 hours for bupivacaine). For patients that are allergic to amide anesthetics, an ester anesthetic, such as procaine, can be used.

In conclusion, it is safe to use epinephrine in digital nerve blocks in the majority of patients. For patients with peripheral vascular disease, caution should be used prior to any block.

## KEY POINTS

- Epinephrine in digital blocks can result in a faster onset and prolonged duration anesthesia.
- The data cited to avoid use of epinephrine in digital blocks were based



on older case reports that had more plausible reasons for digital ischemia.

- Epinephrine can lead to a transient vasoconstriction of digital that has no long-term complications.
- Use caution with epinephrine for digital blocks in patients with peripheral vascular disease or Raynaud syndrome.

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## WHEN ARE PROPHYLACTIC ANTIBIOTICS INDICATED FOR WOUNDS?

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DALE COTTON, MD

The foundation of good wound care includes irrigation, exploration, and selective closure. The decision to prescribe prophylactic antibiotics is an important component of the comprehensive management of acute wounds. Unfortunately, there is limited research to guide clinicians in prescribing prophylactic antibiotics for acute wounds. As a result, clinical decisions are often based on personal experience and common practice patterns. Overall, the incidence of wound infection ranges from 4% to 6%, with the majority caused by skin flora such as *Staphylococcus aureus* and *Streptococcus pyogenes*. The decision to prescribe antibiotics should be based on the likelihood that infection will develop, the consequences of infection, and the adverse effects of antibiotics in the individual patient.

Patient and wound-specific factors should be considered when determining the likelihood of the wound to develop infection. This represents the most complex part of risk assessment and is the area most reliant on clinician judgment. Patient factors associated with an increased risk of wound infection include diabetes, vascular insufficiency, increased age, obesity, renal failure, and immunosuppression (e.g., corticosteroid use, HIV). Wound factors include distal location, prolonged time since injury, presence of crushed or macerated tissue, and contamination of a wound. Unfortunately, no strong data exist to indicate that prophylactic antibiotics prevent infection in these settings. Shared decision making, good communication with the patient regarding the treatment and follow-up plan, and meticulous documentation are important in these scenarios.

Healthy patients with clean, simple wounds do not benefit from prophylactic antibiotics. Wounds involving deep structures (e.g., joints) or associated with fractures are orthopedic emergencies and require specialist consultation to assist with treatment. These wounds are at substantial time-dependent risk of infection; thus, antibiotic prophylaxis should not be delayed. Additional deep structure injuries that have a higher risk of infection and should receive prophylactic antibiotics include extensor tendon injuries or ear wounds with exposed cartilage.

Intraoral lacerations frequently become infected. Limited data suggest that antibiotic prophylaxis is beneficial for intraoral wounds that extend from the intraoral cavity to the external skin surface, are larger than 1 cm or gaping, or are full thickness. Similar to dental infections, penicillin or clindamycin are the most common antibiotics used for prophylaxis of intraoral wounds.

Mammalian bite wounds are at substantial risk of infection due to the mechanism of injury (puncture and crush), the wound location (e.g., hands), and the inoculation of multiple organisms. Dog bite wounds to the distal extremities and human bite wounds benefit from antibiotic prophylaxis. For other bite wounds, the evidence is less clear. Carefully consider patient and wound factors in the decision to prescribe prophylactic antibiotics. Antibiotics with broad-spectrum activity, such as amoxicillin with clavulanic acid, are commonly used for these wounds.

Environmental exposures pose unique infectious risks. Freshwater injuries are associated with *Aeromonas* infection, whereas saltwater wounds are associated with *Vibrio vulnificus* infection. Both gram-negative organisms can cause aggressive infections and may not be covered by antibiotics that simply target skin flora. Though these infections are uncommon, it may be prudent to provide antibiotics against these organisms if water exposure occurred. Soil contamination is another risk factor for infection. These wounds are at risk of infection from *Clostridium perfringens*, a gram-positive anaerobe that is the most common cause of gas gangrene. Penicillin is appropriate prophylactic therapy when considering *Clostridia* infection. Puncture wounds can introduce organisms into deep subcutaneous tissues and should be considered for antibiotic prophylaxis. There is an association of *Pseudomonas aeruginosa* infection with puncture wounds through the rubber soles of athletic shoes. Ciprofloxacin is traditionally used for antibiotic prophylaxis of puncture wounds through athletic shoes.

Patients at risk for infective endocarditis (IE) can be challenging. At present, no recommendations exist for wound antibiotic prophylaxis for this

patient population. The most recent update to the American Heart Association guidelines for prevention of IE does not mention antibiotic prophylaxis for wounds that have no signs of infection. Similarly, the American Academy of Orthopedic Surgeons (AAOS) has no recommendation for wound prophylaxis to prevent prosthetic joint infections (PJI). The AAOS has recommended against antibiotic prophylaxis for dental procedures, as there is no evidence it prevents PJI ([Table 347.1](#)).

**TABLE 347.1 ANTIBIOTIC PROPHYLAXIS SUMMARY**

| <b>NATURE OF WOUND</b>                          | <b>ANTIBIOTIC PROPHYLAXIS</b> | <b>TYPICAL ORGANISMS<sup>a</sup></b> | <b>EXAMPLE REGIMEN<sup>b</sup></b>        |
|---|-------------------------------|--------------------------------------|---|
| Uncomplicated wound                             | No                            | G+                                   | N/A                                       |
| Complex wound or infectious risk factors        | Consider                      | G+                                   | Cephalexin                                |
| Wounds involving deep structures                | Yes                           | G+                                   | Varies                                    |
| Intraoral wounds                                | Yes                           | G+, A                                | Penicillin, clindamycin                   |
| Mammalian bite                                  | Yes                           | G+, G-, A                            | Amoxicillin with clavulanic acid          |
| Environmental (soil)                            | Consider                      | G+, G-, A                            | Amoxicillin with clavulanic acid          |
| Puncture mechanism                              | Consider                      | G+, pseudomonas                      | Varies                                    |
| Water (salt/fresh)                              | Consider                      | Vibrio/aeromonas                     | Doxycycline/trimethoprim-sulfamethoxazole |
| Seeding risk (endocarditis or prosthetic joint) | No                            | G+                                   | N/A                                       |

<sup>a</sup>G+, gram-positive; G-, gram-negative; A, anaerobes.

<sup>b</sup>Prophylactic courses range from 3 to 5 days, no optimal duration has been determined.

## KEY POINTS

- Antibiotic prophylaxis is no substitute for good wound care.
- Healthy patients with clean, simple wounds do not benefit from prophylactic antibiotics.
- Consider patient and wound factors when determining the need for prophylactic antibiotics.
- Wounds associated with fractures require urgent antibiotic prophylaxis.

- Prophylaxis for IE is not beneficial for simple wounds.

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# **Do Not Miss A Foreign Body In A Wound**

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**JASON W. WILSON, MD, MA AND CONSTANTINO DIAZ,  
MD**

Wounds account for ~5% of all emergency department (ED) visits. These injuries are at high risk of contamination and retained foreign body. Retained foreign bodies predispose to infection and can migrate and cause injury to nearby critical structures. It is crucial to identify and remove, if possible, a foreign body in order to improve patient outcome. Importantly, improper wound care and the failure to identify a foreign body account for a substantial number of legal claims against emergency physicians (EPs). A recent review of closed medical malpractice claims demonstrated that retained foreign bodies and contaminated wounds represented 11% to 20% of successful litigation against EPs.

The most common foreign bodies found in wounds are glass, wood, bone, teeth, bullets, metal, gravel, shell, rock, and plastic. Each object has properties that affect the ability to detect them using various imaging modalities. Often, foreign bodies are defined as radiopaque or radiolucent. However, recent literature has shown that these are relative terms and depend on the depth of the foreign body within the wound.

Imaging modalities used for the detection of foreign bodies include plain films (XR), ultrasound (US), computed tomography (CT), and magnetic resonance imaging (MRI). Recently, MRI has been shown to have decreased sensitivity in the detection of foreign bodies due to artifact created by the foreign material. A common theme in successfully litigated cases of missed wound foreign bodies is the failure to obtain diagnostic imaging during the initial ED visit.

An XR should be obtained in any patient when a foreign body is suspected, especially glass. Glass is often apparent on an XR. Be sure to order multiple views of the wound area. When multiple views are obtained, XR can detect a glass fragment >2 mm over 99% of the time. Most metals, bone, and select plastic materials are also detectable on XR. Thin pieces of aluminum, however, may be missed by XR since the anatomic number is close to that of the surrounding tissues. CT should be considered when a foreign body is suspected but not seen on XR. CT should also be considered when the foreign body is deep, close to critical anatomic structures, or when surgery is planned for removal.

US is a noninvasive and cost-efficient approach for the detection and removal of foreign bodies. In fact, US is very sensitive for the identification of wood fragments that are larger than 5 mm. If a patient presents soon after injury, wood can appear bright and echogenic on US. If a patient presents more than 24 hours after injury, a hypoechoic rim is typically seen around the wood fragment. Glass and metal foreign bodies can also be detected using US and appear as echogenic structures with reverberation artifact. Use the high-frequency linear US transducer (10 Hz) in an “in-plane” orientation to achieve the highest likelihood for foreign body detection. The local administration of lidocaine may also enhance the appearance of the foreign body. In addition, the use of a water bath can improve sound wave conduction and help identify soft tissue foreign bodies.

In some patients, a foreign body might be highly suspected but not visualized on diagnostic imaging or direct visualization of the wound. In addition, a foreign body may be located in a precarious position where the risks of removal might outweigh the benefit. In these situations, the patient should be counseled regarding the possibility of a infection and asked to return within 48 hours for a wound check or sooner if fever, erythema, purulent discharge, and worsening pain develops. While antibiotics are not prescribed for patients with a simple laceration, those with retained foreign bodies are at higher risk of infection and may warrant prophylactic antibiotic therapy.

## KEY POINTS

- A retained foreign body can lead to poor patient outcomes and represents a high medicolegal risk for the provider.
- An XR with multiple views should be ordered when there is concern for a retained foreign body.

- XRs readily identify retained glass if the fragment is >2 mm in size.
- CT and US are additional diagnostic imaging modalities that can be used to evaluate the presence of a foreign body.
- Antibiotic prophylaxis may be indicated in patients with retained foreign bodies when the object cannot be removed.

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# KNOW HOW TO TREAT MAMMALIAN BITES

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**CHRISTOPHER I. DOTY, MD, FAAEM, FACEP**

Patients seeking treatment for a mammalian bite is a common occurrence in the emergency department (ED) and is estimated to account for 1 million ED visits each year in the United States. The true incidence of mammalian bites is unknown, as many patients do not seek treatment for injuries sustained from known animals (i.e., pets and neighborhood animals). In addition, many states do not have mandatory reporting of mammalian bites. It is estimated that only 50% of patients suffering a mammalian bite present to the ED for treatment.

The vast majority of bites seen in the ED are from canines. Feline bites account for <10% of visits. It is believed that feline bites are significantly underreported because most of these bites are from the patient's pet. Canine and feline injuries are often bites to the hands or arms and rarely cause significant tissue destruction. Larger breeds, particularly large terrier breeds and canines trained as law enforcement animals, can cause extensive soft tissue destruction, fractures, and crush injuries. Feline bites are more likely to cause puncture wounds, while only a third of canine bites are puncture wounds. A puncture wound should prompt the provider to increase the amount of fluid used for wound irrigation.

Infection is nearly universal with mammalian bites, as the mammal's mouth is teeming with bacteria. However, most of these infections heal well without antibiotics or debridement. Studies have shown a wide variation in infection rates ranging from 1.44% to 30%. It is important to remember that most ED patients present due to bites from a stray canine, bites with significant soft tissue injury, or bites that are already clinically infected. Cat bites that present to the ED are more likely to be infected upon presentation.

Talen et al. showed that both canine and feline bites are polymicrobial, with staphylococcus, streptococcus, and pasteurella being the most predominant organisms.

Not all mammalian bites require treatment with antibiotics. However, recent meta-analyses and systematic reviews have demonstrated that prophylactic antibiotics do show benefit in select circumstances. Mammalian bites to the distal extremities, particularly the hand, show a significant reduction in clinically relevant infections if treated with a proper antibiotic regimen. Furthermore, bites sustained from humans showed a decreased risk of clinically relevant infection if treated with antibiotics as well. One study demonstrated a strong trend for decreased infections in feline bite wounds if treated with antibiotics, but the data did not reach statistical significance. It is critical to remember that all of these meta-analyses and systematic reviews are based on several small, flawed studies. Better evidence for the utility of antibiotics in these patients does not presently exist. The most recent evidence supports treating all mammalian bites to the distal extremities and all bites from humans. Patients with select clinical characteristics of high-risk wounds should also be considered for antibiotics. These characteristics are listed in [Table 349.1](#). Importantly, antibiotics should cover  $\beta$ -lactamase-producing bacteria.

**TABLE 349.1 CLINICAL CHARACTERISTICS OF HIGHER-RISK BITE WOUNDS**

|                                 |                                |
|---------------------------------|--------------------------------|
| Wound infection already present | Diabetes                       |
| Location on a distal extremity  | Immunocompromised state        |
| Wounds over 12 hours old        | Preexisting edema              |
| Age over 50 years               | Vascular disease               |
| Asplenia                        | Significant tissue destruction |
| Chronic ethanol use             |                                |

All mammalian bites should receive analgesia, copious irrigation, assessment for tetanus prophylaxis, and potential rabies treatment. Complex wounds should receive higher amounts of irrigation. It is recommended that mammalian bite wounds should not be closed primarily, but this is sometimes impractical with large or cosmetically highly visible wounds.

## FIGHT BITES

It is critical for emergency physicians to realize that tooth injuries to the metacarpal phalangeal (MCP) joint or “fight bites” should be treated more aggressively than other human bites. These injuries occur when the patient strikes another person in the mouth with a closed fist. The clinical concern is the inoculation of the superficial tendon and sheath as it passes over the dorsal, extensor surface of the MCP joint. A closed fist has the extensor tendons at maximal length. The damaged and contaminated tendon then retracts up the sheath carrying saliva and bacteria with it. A small MCP skin laceration can appear innocuous but a significant infection may be evolving. A suggestive history in the presence of even a small MCP skin defect should prompt a thorough evaluation. “Fight bites” have an infection rate up to 75%. Approximately 60% have deep structure involvement including tendon injury, joint involvement, and fractures. These injuries should be washed out in the ED or in the operating room and should be seen by a hand surgeon emergently.

## KEY POINTS

- The more complex the wound, the more it should be irrigated.
- Antibiotics should be reserved for bite wounds that occur on the distal extremities and any bites by humans.
- All lacerations at the MCP joint should be considered a “fight bite.”
- Consider rabies prophylaxis in all mammalian bites.
- Proper antibiotic coverage for mammalian bites should include medications to cover  $\beta$ -lactamase-producing bacteria.

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## IS THAT SKIN LESION AN INFECTION OR AN ENVENOMATION?

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Patients frequently present to the emergency department (ED) with a chief complaint of “spider bite.” The likelihood that a skin lesion is due to a spider often depends on where the patient lives. The majority of lesions attributed to spiders are actually cutaneous abscesses, cellulitis, or other soft tissue conditions such as vasculitis, neoplasms, chemical burns, and venous stasis ulcers. The challenge for the emergency provider (EP) is to recognize the patient with a true spider bite, as the management is very different from the treatment of more common soft tissue conditions.

There are an estimated 4,000 spider species in the United States (US). Importantly, only several species cause clinically significant reactions in humans. The most important are *Latrodectus mactans*, the black widow spider, and *Loxosceles reclusa*, the brown recluse spider. The black widow spider is found throughout the US. Envenomation produces systemic toxicity rather than significant cutaneous findings. As a result, it is uncommon for the EP to confuse skin infections with black widow envenomation. In contrast, *Loxosceles* envenomation often produces significant cutaneous manifestations with or without systemic involvement. The remainder of this chapter will focus on the brown recluse spider bite.

Distinguishing a brown recluse spider bite from a cutaneous infection can be challenging. Brown recluse spiders are found in the south central US, especially in Arkansas, Missouri, and Kansas. Less common recluse species (i.e., *L. deserta*, *L. arizonica*, *L. blanda*, *L. apachea*, *L. devia*) are confined to the southwest. Patients living outside of these endemic areas are unlikely to

have a brown recluse bite. For patients who live in endemic regions, it is important to accurately identify the spider, if possible. *L. reclusa* vary in size; however, the body is usually 2 to 3 cm long. The eight legs may each extend up to 20 mm. The distinguishing feature of a brown recluse spider is the violin-shaped marking on the dorsal cephalothorax. Brown recluse spiders also possess six eyes, whereas most other spiders have eight eyes. It is important to remember, however, that proximity to a brown recluse spider does not mean that the spider is responsible for the patient's signs and symptoms.

Brown recluse spider bites present with local findings that follow a predictable course. The bite itself causes little to no pain. A nonpurulent blister soon appears, followed by surrounding erythema that spreads in a gravitational pattern. Over the next 2 to 3 days, wound discomfort, induration, and edema develop. An area of central necrosis appears in ~50% of patients between days 2 and 4. Concentric rings of ischemia and erythema, resulting in a "red, white, and blue" appearance, often surround the necrotic area. Over the next few days, an eschar develops over the necrotic area, which typically heals over a period of weeks. Lymphangitis and purulent drainage are not characteristic of brown recluse bites. Importantly, incision or excision of a brown recluse bite is associated with increased pain, delayed wound healing, and a higher incidence of infection.

In contrast to a brown recluse spider bite, cellulitis is erythematous and often exquisitely tender. Lymphangitis may also be seen. Cutaneous abscesses are generally fluctuant and may spontaneously drain purulent material. Ultrasound is useful for distinguishing abscess from cellulitis. Severe soft tissue infections may appear violaceous, have gas or crepitus in the subcutaneous tissue, and have signs of systemic toxicity.

On occasion, brown recluse envenomation may cause systemic manifestations such as fever, myalgias, nausea, and vomiting. Systemic loxoscelism is characterized by a rapidly progressive scarlatiniform rash, abdominal pain and tenderness, and hemolysis. Children are especially susceptible to the effects of this hemolysis, which can lead to profound anemia, cardiovascular collapse, and death. Systemic loxoscelism must be treated aggressively.

Laboratory tests are not helpful in confirming the diagnosis of a brown recluse bite. In cases of systemic loxoscelism, anemia, thrombocytopenia, increased urinary hemoglobin, decreased haptoglobin, renal dysfunction, hepatic dysfunction, and coagulation abnormalities may be noted.

It is essential for the EP to be able to identify a brown recluse spider bite. While most bite wounds will heal spontaneously, 15% to 20% may require

skin grafting several weeks after the envenomation. Antibiotics are unnecessary for a brown recluse bite. Evidence for the use of the dapsone in brown recluse envenomation is scant. In addition, dapsone has significant side effects that include methemoglobinemia, cholestatic jaundice, and hypersensitivity reactions.

## KEY POINTS

- Brown recluse spider bites are restricted to select geographical regions of the United States.
- A brown recluse spider bite presents with a small, nonpurulent blister, which develops into a central area of necrosis surrounded by concentric rings of ischemia and erythema.
- The area of central necrosis forms an eschar within the first week and typically heals spontaneously over the ensuing weeks.
- Brown recluse spider bites do not produce fluctuant, purulent lesions.
- Systemic loxoscelism is characterized by a rapidly progressive rash, abdominal pain, and hemolysis.

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# **KNOW HOW TO TREAT SNAKE BITES**

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There are ~9,000 snakebites annually in the United States (US). Although snakebites are an uncommon emergency department (ED) complaint, it is imperative to know how to properly treat snakebite victims to avoid unnecessary morbidity and mortality.

There are more than 100 species of snakes indigenous to the US. Thankfully, only about 20 species are venomous snakes. Most of these venomous snakes belong to the Viperidae (sub-family Crotalinae) or Elapidae family. Crotaline snakes (i.e., rattlesnake, copperhead, moccasin) are commonly called “pit vipers” and account for nearly all the venomous snakebites in the US. Pit vipers have a triangular head, elliptical pupils, and a pair of small, heat-sensing pits between each eye and nostril. Up to 25% of pit viper bites do not result in envenomation, commonly referred to as a “dry” bite. Coral snakes are the most well-known member of the Elapidae family of venomous snakes. These are brightly colored with black, yellow, and red rings. The phrase “red on yellow, kill a fellow; red on black, venom lack” is often used to differentiate coral snakes from the nonvenomous king snake. There are no venomous snakes indigenous to Alaska, Hawaii, or Maine.

The majority of snakebites are limited to the subcutaneous tissues. Only rarely does a bite reach deeper tissues. When venom is injected, it travels along the lymphatic system and superficial veins to reach the central circulation. Rapidly fatal envenomation can occur if venom is injected directly into a vessel. Crotaline venom causes direct cell damage, capillary

leak, a consumptive coagulopathy and, to a lesser extent, neurotoxicity. Clinical symptoms range from local symptoms to life-threatening systemic reactions. Local symptoms include pain, erythema, edema, or ecchymosis at the bite site. Systemic symptoms include nausea, vomiting, lethargy, weakness, and perioral and extremity paresthesias. Hypotension, tachypnea, respiratory distress, tachycardia, altered mental status, renal failure, and death may also be seen in severe reactions. Rattlesnake bites often result in a consumptive coagulopathy, as manifested by elevations in the international normalized ratio (INR), prothrombin time, and fibrin degradation products. Thrombocytopenia ( $<20,000$  cells/mm<sup>3</sup>) is also characteristic.

Coral snake venom is an  $\alpha$ -neurotoxin. This toxin blocks postsynaptic nicotinic acetylcholine receptors at the neuromuscular junction. Fang marks are often difficult to see, and there are often minimal local findings. Immediate symptoms may include numbness at the bite site. Cranial nerve abnormalities can be seen and include ptosis, dysarthria, and dysphagia. Respiratory paralysis can also develop. Systemic symptoms can be delayed for up to 12 hours but are difficult to reverse once present.

Prehospital treatment of the snakebite victim includes immobilization of the affected body part, avoidance of excessive activity, and transportation to a local hospital. The affected limb should be maintained at the level of the heart. All jewelry and constrictive clothing should be removed. Arterial tourniquets, aggressive wound incisions, and ice are no longer recommended. A wide band proximal to the bite site compressing only superficial vessels may be applied and left in place until the patient reaches definitive medical care. Immobilization and compression have been shown to be helpful in Elapidae snakebites but remain unproven in pit viper envenomation. The use of a venom extractor has been shown to increase local tissue damage and is not recommended.

In the ED, wound care should be performed, with tetanus updated if applicable. Patients should undergo an evaluation for hematologic, neurologic, renal, and cardiovascular abnormalities. Blood work should be drawn from an unaffected limb and sent for a complete blood count, coagulation studies, electrolytes, blood urea nitrogen, serum creatinine, and creatine phosphokinase. Additional blood work may include a type and cross-match, fibrinogen, fibrin split products, and bleeding time. A urinalysis and electrocardiogram should also be obtained. Poison center consultation should also be obtained. An x-ray can be obtained if there is concern over a retained fang. Prophylactic antibiotics are not recommended.

Patients with pit viper bites should be observed for approximately 8 to 12 hours. If there are no signs of envenomation, the patient may be discharged



home. When envenomation does occur, the edge of the swelling should be demarcated with a pen, and the circumference of the extremity should be measured every 15 to 30 minutes. If there is no progression of swelling and no coagulopathy develops on serial lab values, the patient can be discharged home.

In contrast to pit viper bites, patients with coral snake bites should be observed for at least 24 hours, even without definitive signs of envenomation. If envenomation is suspected, they should be treated immediately with antivenin, since symptoms are irreversible.

The antivenin Crotalidae Polyvalent Immune Fab (Ovine) (CroFab; BTG International, West Conshohocken, PA) has supplanted the older horse-derived Antivenin (Crotalidae) Polyvalent (ACP) for the treatment of crotaline envenomation. The Ovine formulation is as effective as the horse-derived antivenom, but with a reduced risk of allergic reaction. Antivenin should ideally be administered within 4 hours of envenomation but can be effective for up to 24 hours following the bite. Indications for antivenin include progressive swelling, a coagulation abnormality, or development of systemic effects. The initial dose of antivenin is 4 to 6 vials given over 1 hour. If symptoms are controlled, the patient should receive two vials at times 6, 12, and 18 hours. If initial control is not achieved, the patient should receive an additional 4 to 6 vials. Be prepared to treat anaphylaxis in any patient receiving antivenin.

For any coral snake bite, regardless of symptoms, *Micrurus fulvius* antivenin (equine) is recommended. This antivenin has no activity against the Sonoran, Arizona, coral snake. Supplies of this antivenom will soon be exhausted, as the manufacturer stopped production in 2003. There is currently a study of a F(ab')<sub>2</sub> antivenom in Phase 3 clinical trials. If an exotic snakebite occurs, local zoo or poison center experts should be contacted.

## KEY POINTS

- Monitor patients closely for systemic and hematologic effects.
- “Red on yellow, kill a fellow; red on black, venom lack” can be used to differentiate a coral snake from a king snake.
- Coral snake bites require antivenin even without signs of envenomation.
- Antivenin may need to be readministered several times until there is control of symptoms.
- Have epinephrine and antihistamine ready when administering

antivenin in case of an anaphylactic reaction.

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# **EYELID LACERATIONS: WHEN TO REPAIR AND WHEN TO REFER**

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It is important for the emergency provider (EP) to know how to manage an eyelid laceration. Poorly managed lid lacerations often lead to eyelid and tear duct dysfunction, a source of significant patient discomfort as well as liability for the EP.

The eyelid is composed of several layers including the skin, orbicularis muscle, and the orbicularis septum. The orbicularis septum is a fibrous material that separates the superficial eyelid structures from the deeper structures. Orbital fat and the levator palpebrae muscle lie beneath the orbicularis septum. The levator muscle is important for eyelid function and inserts at the tarsal plate along the eyelid margin. Puncta are located in the medial eyelid margins and connect via canaliculi to the lacrimal duct system.

Eyelid laceration repair should occur only after the eye has been thoroughly evaluated for injury. Document the visual acuity and complete a thorough eye exam. If globe rupture is suspected, laceration repair should not be performed, as increased pressure on the globe can cause additional injury. The history of the incident is important in assessing the complexity of the laceration. Important historical features include the mechanism of injury (i.e., blunt vs. penetrating), time of injury, presence of foreign bodies, and any prior ocular history. Retained foreign bodies can lead to infection and discomfort. Plain radiographs, ultrasound, or computed tomography should be obtained if there is concern for a retained foreign body. Importantly, the eyelid is highly vascular, and laceration repair can be deferred up to 36 hours if needed.

It is important to determine whether the eyelid laceration is simple or

complex. Features that determine simple or complex include location, depth, and size.

- 1) Location. Any laceration that involves the lid margin or communicates with the lacrimal duct system is complex and should be referred to a specialist for repair. Placing a drop of fluorescein into the eye and then examining the laceration with the blue light can evaluate the lacrimal duct system. If fluorescein is detected within the wound, then it communicates with the lacrimal duct system.
- 2) Depth. The presence of fat within an eyelid laceration indicates that deeper structures, like the levator muscle, may be injured. Ptosis is another key exam finding to suggest that the levator muscle is compromised. These lacerations should be considered complex and referred to a specialist for repair.
- 3) Size. Avulsion injuries should be considered complex lacerations and referred for repair, as improper repair can cause wound tension that affects lid function. Small lacerations that are superficial and less than 25% of the lid width can be left to heal by secondary intention. Tissue adhesive is an option for small lacerations, but care should be taken to prevent the adhesive from entering the eye.

The EP can repair simple lacerations. A small, 6-0 or 7-0 nylon suture, or rapidly absorbing suture, may be used with a simple interrupted technique. The ends of the suture should be cut short to avoid irritating the eye. If the wound is near the lid margin, then the sutures most proximal to the margin should be buried. An alternative is to leave the ends of the proximal suture long and incorporate them into the next suture tie, effectively tying them down and keeping them away from the eye. Puncturing the eye with the suture needle can occur. One technique to avoid globe injury is to leave the first suture ends long. The provider then uses the ends to gently pull traction and lift the lid away from the globe. Another trick is to insert an ocular anesthetic and then a Morgan's lens, which can act to shield the globe during repair.

Eyelid lacerations are a common problem facing the EP. The first step is to evaluate the eye globe injury. Complex lacerations involving the eyelid margin, lacrimal duct, or levator muscle should be referred to a specialist for repair. Repair can be delayed up to 36 hours. Simple wounds can be repaired using a small suture with an interrupted technique. Care must be taken to avoid iatrogenic globe injury.

## KEY POINTS

- Document a visual acuity and perform a thorough eye exam prior to lid laceration repair.
- The presence of fat in the laceration indicates penetration of the orbicularis septum.
- Any laceration that involves the lid margin or lacrimal system should be referred for repair.
- If fluorescein appears in the wound after placing in the eye, it suggests violation of the lacrimal system.
- A Morgan lens can be used to protect the globe from iatrogenic injury during laceration repair.

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## WEB SITES

<http://lacerationrepair.com/special-situations/lacerations-around-the-eye/>

## EAR INJURIES AND LACERATIONS

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Ear injuries generally occur as a result of blunt trauma or mammalian bites. Appropriate management of these injuries is critical to prevent serious complications. The irregular contour of the ear, blood supply, and underlying cartilaginous structures can make these injuries difficult to manage. Unfortunately, randomized-controlled trials are lacking to guide the care of these injuries in the emergency department (ED).

One of the first steps in evaluating an ear injury is to determine whether the cartilage has been injured or exposed. When cartilage is involved, injuries should be classified as complete or incomplete avulsions. Plastic surgery or otolaryngology (ENT) should be consulted emergently for complete avulsion injuries. Avulsed tissue should be reattached as quickly as possible. Detached tissue can be cleaned in cold saline, but this is best done in consultation with the specialist. Consultation should also occur for partial avulsion injuries with very small pedicles of tissue.

The decision to repair external ear injuries in the ED is dependent on the extent of tissue loss, the time elapsed since the injury, and any associated injuries. Proper repair of ear injuries and lacerations requires appropriate anesthesia. Small lacerations can be anesthetized with local infiltration of lidocaine. It has been traditionally taught to avoid the use of epinephrine in this region. However, hemostasis is important for repair and prevention of an auricular hematoma. There is an evidence that epinephrine does not lead to complications when used for repair of ear injuries. A regional auricular block may be required for large or complicated lacerations. Children and uncooperative patients may require procedural sedation for proper repair.

The emergency provider (EP) can consider primary closure if the injured

portion of the ear is on a wide pedicle and demonstrates good distal capillary refill. The perichondrium and subcutaneous layers should be sutured closed with absorbable sutures. The skin should be approximated with 5-0 or 6-0 nonabsorbable sutures (i.e., nylon or polypropylene). Rapid absorbing 5-0 or 6-0 sutures may be acceptable in children when there is concern for difficult removal. Use the contralateral ear for comparison to help guide the repair of the affected ear. Patients should receive specialist follow-up after primary closure, as the repair can be revised if the cosmetic outcome is not satisfactory.

Allowing ear wounds to heal by secondary intention may be appropriate in select patients, namely diabetic patients, immunocompromised patients, or those with heavily contaminated wounds. Small wounds to the concave portions of the auricle (conchal bowl and antihelix) heal particularly well by secondary intention, provided the surrounding ear is intact to provide structural support. The wound requires copious irrigation even when primary closure is not performed. Additionally, the wound should be covered with antibacterial ointment and any crusting removed. Exposed cartilage should be avoided, as the overlying skin provides its vascular supply. Patients who do not have their wounds closed in the ED should be referred for follow-up within 1 to 2 days with an ENT or plastic surgeon.

The primary complications of ear injuries are infection, auricular hematoma, and poor cosmetic appearance. Consider antibiotics for patients with diabetes, immunocompromising conditions, those receiving chemotherapy or corticosteroids, or if the patient sustained their injury from a human or animal bite. The EP should also consider antibiotics for contaminated or macerated injuries. Auricular hematomas are usually the result of blunt trauma to the auricle. The skin adheres to the perichondrium, which supplies blood to the cartilage, and the unique anatomy of the ear does not allow for significant expansion of the subcutaneous tissue. Blood accumulates in the subperichondrial space and disrupts blood supply to the underlying cartilage. To prevent formation of an auricular hematoma, consider placing a pressure dressing after wound closure and give patients strict instructions to return for any signs of swelling. Auricular hematomas should be drained as soon as possible to prevent the development of fibrocartilaginous overgrowth and deformity of the ear. Two approaches have been described in the literature to aid with the drainage of these hematomas. Generally, smaller hematomas can be drained with needle aspiration, whereas larger hematomas should be drained with incision and drainage. Following drainage, patients should be regularly followed for at least 1 week to ensure there is no recurrence. Hematomas more than 7 days old should be referred to a specialist.

## KEY POINTS

- Consult ENT or plastic surgery emergently for complete avulsion injuries.
- Ensure all cartilage is covered with a wet-to-dry dressing following repair. These dressings should be changed daily until follow-up with a specialist.
- Many small lacerations to the concave portions of the ear can be allowed to close by secondary intention provided the supporting peripheral structure of the ear is intact.
- Antibiotics are recommended in bite wounds and should be strongly considered in immunocompromised patients.
- All patients with ear lacerations closed in the ED should be referred to ENT or plastic surgery for follow-up.

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# **KNOW WHICH WOUNDS TO CLOSE...AND WHICH ONES TO LEAVE OPEN**

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Several million patients present annually to the emergency department (ED) with an acute wound. For these patients, the emergency provider (EP) must decide between primary closure, delayed closure, or allow the wound to heal by secondary intention. Both patient and wound factors are critical in making this decision. In addition, it is important to assess patient concerns, such as functional outcome, the potential for painful procedures, and the final cosmetic appearance. For the EP, it is important to prevent the loss of function, decrease the risk of infection, and achieve acceptable cosmetic outcomes. The fundamentals of ED wound management include an accurate history of present illness (HPI), assessment of patient comorbidities, allergies, and tetanus status and a thorough wound assessment that includes debridement of devitalized tissue and removal of foreign bodies.

An important element of the HPI is a determination of the amount of time that has elapsed since the injury. This time frame has classically been referred to as the “golden period,” after which the rate of infection significantly increases. In 1898, Paul Leopold Friedrich was the first to describe a 6-hour “golden period” for wound closure, based on the data derived from a guinea pig model. At present, there is no high-quality data to suggest an ideal time for primary closure, beyond which there is an increased risk of infection. Another important component in the assessment of wound infection is location. Wounds above the clavicles generally have a

lower risk of infection compared with extremity wounds. A history of diabetes, immunosuppression, increased age, large wounds, and the presence of contamination or a foreign body increase the risk for infection. Thus, a clean facial wound can often be closed primarily, even up to a day or more from the time of injury. In contrast, a contaminated foot laceration in an elderly diabetic patient is likely a poor choice for primary closure, even just a few hours following the injury.

Mammalian bite wounds have a higher risk of wound infection compared to nonbite wounds. Dogs, cats, and humans most commonly cause bite wounds. Wound characteristics vary with the type of bite. Dog bites are more likely to result in lacerations, avulsions, and crush injuries, with or without fractures, due to the force generated during the bite. Cats create deep, penetrating puncture wounds that can deliver infectious inoculum deep into subcutaneous tissues. Human bites often involve the fourth or fifth metacarpophalangeal joint of the hand with possible tendon injury. The location of bite wounds is also critical. High-risk wounds involve the extremities, overly joints, or demonstrate deep tissue damage. Human or cat bites that involve the hands or feet have a high risk of infection and should be left open. Facial bite wounds, however, may be closed primarily as the cosmetic benefit often outweighs the risk of infection.

In general, EPs should identify risk factors for wound infection on a case-by-case basis, as wounds at high risk of infection should be considered for delayed closure. These include human and cat bites to areas of the body except the face. Dog bite wounds can be closed primarily, except in the case of hand wounds and a delayed presentation from the time of injury. The decision to close or leave open nonbite wounds should consider host and wound factors such as time since wounding, medical comorbidities such as diabetes, immunocompromised states, peripheral vascular disease, increased age, wound contamination, presence of foreign body, anatomic location, and wound size. Grossly contaminated wounds with significant amounts of devitalized tissue should be left open. Regardless of the closure strategy, each wound should be copiously irrigated to decrease the risk of infection. As always, the risks and benefits of pursuing primary versus delayed closure techniques should be discussed with the patient and appropriately documented so that an informed and shared decision can be made. With no clear guidelines, clinical judgment is the key in weighing the benefits of wound closure against the risks of infection.

## KEY POINTS

- The history and physical examination should be directed toward an assessment of the overall risk for infection.
- Important elements to the HPI include time since injury, patient comorbidities, anatomic location, wound dimensions, and the presence of foreign material, gross contamination, or devitalized tissue.
- Facial bite wounds can often be closed primarily, as the cosmetic benefit outweighs the risk of infection.
- All wounds should be copiously irrigated.
- Discuss the risks and benefits of wound closure with each patient and appropriately document the conversation.

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## **SECTION XXIV**

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# **CLINICAL PRACTICE AND LEGAL ISSUES**

# CONSULT COMMUNICATIONS: OPTIMAL COMMUNICATIONS WITH CONSULTANTS

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**HUGH F. HILL III, MD, JD, FACEP, FCLM**

Ask why we request consultations, ask what are the common elements of consults, and you will know how to avoid common errors. In other words, *break it down!*

We call consultants for:

- Help with diagnosis
- Workup and treatment advice
- Specific procedural (medical and administrative) assistance
- To assure family and patients of our thoroughness and accuracy
- To coordinate and share responsibility for care

Once the need or perhaps opportunity for consultation is realized, the linguistic elements of every consultation align in consistent order:

- Contact
- Communication about the patient
- The question(s) or action requested
- Assent
- Response and report
- The requestor's closure

## HELP WITH DIAGNOSIS

Our requests are less often for another specialist's thoughts about a diagnosis than those requested in the office outpatient or in-hospital setting. But we still use our colleagues' help occasionally in this way, for example, what's this rash, could I be missing any other reasons for this persistent hypotension? These requests require more complete information inclusion. We avoid bias in the presentation of that information; the consultant is less likely to pick up something we missed if we lead her too much.

## **WORKUP AND TREATMENT ADVICE**

We often ask for comments about preadmission studies or choice of imaging. While these communications can be brief, we profit from disciplining ourselves to think of them as requests for consultation. Talking with the team that will be caring for the patient after admission can only help. It is not an abrogation of professional autonomy or status for us to modify our work to better fit with what will happen next, when we no longer have control, where it's reasonable.

## **SPECIFIC PROCEDURAL (MEDICAL AND ADMINISTRATIVE) ASSISTANCE**

We know what must be done, so we call the right specialist to do it. Nonetheless, it behooves us to acknowledge their independent judgment. Good hospital interstaff relations and ultimately patient care depend on smooth interactions. It's rare that we have to go to the extreme of calling a second same-specialty consultant because the first is making a dangerous choice. Our non-EM colleagues usually don't have a call list: they call whoever they want. If our patients are not getting the support they need from a consultant on the call list, we have to address it through staff mechanisms.

## **TO ASSURE FAMILY AND PATIENTS OF OUR THOROUGHNESS AND ACCURACY**

In some EDs, sophisticated and resource-enabled patients and families are accustomed to self-referring to specialists. They may not even have a primary care generalist. Even if all you do is talk with their choice for follow-up, they can be reassured. But continue to think of it as a consult, requiring elements of communication, specificity, and loop closing.

# **TO COORDINATE AND SHARE RESPONSIBILITY FOR CARE**

Although not usually recognized as a consultation, calling the primary care provider before the patient leaves the ED can be. For example, imagine that she says, “I know this patient and this is atypical. Please admit him.” Even if you don’t admit, your documentation burden just escalated. The most likely source of criticism of your care in the ED remains the next provider to see your patient. Most do not, but even a raised eyebrow can start a patient or family thinking about litigation. The follow-up doctor who has “bought into” your care plan is much less likely to point a finger or even question your care.

Once you’ve decided you need a consult, a formulaic approach can help. (Please note: Starting the process of seeking consultation establishes the necessity of completing it. If the physician you are calling doesn’t respond, you can be said to have a responsibility to persevere or call someone else. Desisting requires documentation of why.)

## **THE ELEMENTS OF CONSULTATION**

### ***Contact***

If during office hours, be respectful of your consultant’s other responsibilities. If time allows, ask her staff to call you back. If the consultant is not working, either make the call yourself or be very available if the unit secretary or other personnel make the call. Introduce yourself, say where you are calling from, and try to confirm you are talking to the right person. A respectful, “I have you on call tonight; is that correct?” can turn away wrath.

### ***Communication about the Patient***

The range and content of your presentation will alter with circumstances of the case and what you are asking. Don’t edit the information beyond what you would want to know if the situation was reversed. Explain why the datum that doesn’t fit should be discounted, but reveal it.

### ***The Question(s) or Action Requested***

This requires clarity. We need to articulate specifically what we want from

the consultant.

## ***Response and Report***

This is what the consultant does. If she doesn't immediately put in a note, then we have to record what we understood the response to be. When the consultant takes responsibility—"Yes. Admit to my service."—it's obvious. (Note: we still have to judge the reasonableness of the consultant's communicated immediate intentions. Further, our continued responsibility is clearer until the consultant sees the patient.)

## ***The Requestor's Closure***

As with any sequential charting, we risk conflicting statements if the consultant enters documentation after we have put our note in. In some situations, we may go back and read the subsequent entry and add a properly timed addendum after.

Finally, many authors urge that we abstain from "curbside" consults. If you do informally talk with another specialist, play fair. Don't record what you are told as if it were the result of the formal process. If you set up another physician for untoward responsibility, you and your EM colleagues will have more problems than conflict with that one doctor. If you want to chart something, consider, "Called Dr. X and he will F/U."

### **KEY POINTS**

- Be clear in your own mind why you are calling a consultant.
- Communicate what you want from the consultant clearly.
- Avoid bias in your presentation to the consultant.
- Document the time you call and the time of response.
- Close the loop and note the results of the consultation.

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# **TREATING THE PATIENT AND NOT THE DISEASE: TIPS FOR PATIENT SATISFACTION**

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**DYLAN SEAN KELLOGG, MD**

We don't think of emergency medicine as a customer service business. Our patients arrive dying, we stop them from dying. There is some intangible benefit from being compassionate and making patients happy but surely that is not the goal of our profession. Moreover, we often worry about patients who are drug seeking or demanding unreasonable tests.

The reality, of course, is that a large segment of our patient population is not trying to die in front of us, the majority of our patients are not just looking for narcotics, and all have concerns that go beyond a clinical diagnosis. The way we are paid is often tied to satisfaction metrics. Studies have shown that increased patient satisfaction is tied to decreased litigation. In urban environments, where multiple emergency departments (EDs) are within close proximity, having satisfied patients contributes to increased volumes through word-of-mouth advertising and retention of existing patients. More satisfied patients are more likely to be compliant with, and respond to treatment, and are less likely to require follow-up ED visits. Finally, happier patients create a more positive working environment and happier staff.

Five variables make the biggest difference in patient satisfaction scores: technical competence, timeliness of care, empathy, information dispensation, and pain management. Committing to address these variables in your own clinical practice and on a departmental level can make significant improvements in patient satisfaction.

Technical competence is a domain in which emergency physicians excel, and in some studies, it is the most important variable in satisfaction scores. Unfortunately, academic centers can lose satisfaction because of the need to have inexperienced trainees perform technical skills on patients. This can be mitigated by the use of scripting—training providers on how to approach difficult conversations with patients, like trainees’ involvement in care.

Timeliness of care is one of the most commonly cited complaints about ED visits, and boarding and overcrowding are unlikely to disappear any time soon. While there will always be some form of waiting in EDs, there are simple steps that can be taken to improve patient satisfaction. Patients want to be seen by a doctor as soon as possible and the longer they have to wait to do so, the more likely they are to leave. Arranging department flow to decrease door-to-doctor time can have a significant effect on patient satisfaction, and practices like bedside registration, and placing a provider in triage can be effective. If possible, try to keep door-to-doctor times to under 30 minutes. Patients care more about perceived wait times than actual wait times, so shortening perceived waiting also improves satisfaction. Having waits that are shorter than expected, allowing for visitors, providing information on how the department functions and what to expect, and providing frequent updates on care can all improve perceived wait times.

It can take a single negative interaction with a staff member to make the entire visit a poor experience. Dress professionally: patients respond negatively to providers who are dressed casually or appear unkempt. Wearing clean, professional attire is an important (and easy) step to enhance patient satisfaction—providers should consider wearing white lab coats, as at least half of patients respond favorably to them. All personnel should wear prominently displayed ID badges, introduce themselves to patients and family members, and explain their role in the patient’s care. Good bedside manner that includes sitting and listening patiently is a must. Your department should have interpretive resources in place for non-English-speaking patients. Empathy can also be improved with scripting by preparing staff for potentially difficult interactions with patients and family.

Since pain is one of the most common reasons for patients to come to the ED, pain management is an important tool for satisfaction. Scripting and setting realistic pain management expectations play important roles in patient satisfaction. When possible, allow patients to participate in decision-making regarding pain management and institute departmental policies that allow for nurse-initiated pain management prior to patients seeing a provider.

Information dispensation is frequently overestimated by ED providers. During a patient’s stay in the department, frequent updates help to keep

patients informed about their progression (and decrease perceived wait times). Consider instituting a system of ED rounding, wherein patients are given regular timed updates on their care by staff members. The presence of a patient advocate in the department can also help patients understand what is happening. On discharge, provide clear instructions with follow-up and return precautions. Provide patients with your business card so that they can call with additional questions. Multiple studies have demonstrated that a follow-up phone call or e-mail from the ED provider after discharge significantly improves patient satisfaction.

Ultimately, doctors and nurses are in the business of making people feel better. This is especially true in the ED where our average day at work is often our patient's worst day. Patient satisfaction should not be viewed as separate from clinical care. Making our patients feel better by making them feel more welcome in the department, better informed about their care, and more satisfied when they are discharged is just good medicine.

## KEY POINTS

- Dress professionally: ideally wear a white lab coat.
- Institute scripting for difficult situations.
- Keep door-to-doctor time to <30 minutes.
- Implement ED rounding.
- Follow-up with patients after discharge (phone call or e-mail).

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# YOUR PATIENT HAS DIED, NOW FOCUS ON THE FAMILY: HOW TO DELIVER BAD NEWS TO FAMILY MEMBERS

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**DYLAN SEAN KELLOGG, MD**

It's 2 am, you've spent the last 45 minutes attempting to resuscitate a middle-aged man without success. You thank your team and prepare to fill out the appropriate paperwork when one of the registration staff comes up to you. "Doctor, the patient's wife is here."

Most of us had brief instruction on "how to break bad news" during medical school that involved role-playing. This was often before we had any actual clinical exposure and was almost certainly not specific to emergency medicine. During residency, we were expected to start having these conversations. They don't get easier, and when every family reacts differently, how can you tell if you're doing a good job? Moreover, these conversations are often viewed as an afterthought—we've taken care of the patient, now let's inform the family.

This is the wrong approach. Treating a bereaved family member with the same care as you would a patient facilitates both parties in dealing with an incredibly challenging situation and provides better long-term outcomes for the family.

We are extremely systematic in how we approach patient encounters and that same rigor should be used in interactions with family members. Two mnemonics that have been studied as tools to help in bad news conversation are **SPIKES** ([Table 357.1](#)) and **GRIEV\_ING** ([Table 357.2](#)). The first was

originally studied in oncology but has been applied successfully to the emergency department, while the second is less well studied but was designed for emergency department application. Both stress the importance of preparing for the conversation, providing a private, nonclinical space, making sure the appropriate people are present, determining how much is known before you begin speaking, being direct yet empathetic in delivering news, and providing opportunity for questions and follow-up.

**TABLE 357.1 SPIKES**

|                      |  |
|----------------------|--|
| <b>S: Setting up</b> | Privacy, appropriate people present, pager off, sit down                   |
| <b>P: Perception</b> | How much does person know already?   |
| <b>I: Invitation</b> | Make sure person is ready to receive the information you are going to give |
| <b>K: Knowledge</b>  | Warn bad news is coming, use nontechnical words, avoid bluntness           |
| <b>E: Emotions</b>   | Address emotional reaction   |
| <b>S: Summary</b>    | Summarize, help with planning for future if person is ready                |

**TABLE 357.2 GRIEV\_ING**

|                            |  |
|----------------------------|--|
| <b>G: Gather family</b>    | Ensure important people are present  |
| <b>R: Resources</b>        | Get support: chaplain, social worker, supportive family/friends            |
| <b>I: Identify</b>         | Introduce self, identify deceased by name, how much does family know       |
| <b>E: Educate</b>          | Briefly explain events including current state of patient                  |
| <b>V: Verify</b>           | Verify that patient is dead—use words “dead” or “died”                     |
| <b>_: Space</b>            | Give space and time to absorb  |
| <b>I: Inquire</b>          | Inquire if there are questions and answer them                             |
| <b>N: Nuts &amp; Bolts</b> | Offer opportunity to view body; ask about belongings, organ donation, etc. |
| <b>G: Give</b>             | Provide business card, offer to answer later questions                     |

Some tactics bear emphasis. Check your appearance—make sure you aren’t covered in blood. Have a support person present—it isn’t feasible for a physician to remain with family during their visit, and it is inappropriate to leave family alone, so having a chaplain, social worker, or nurse stay with them is helpful. Verify your facts—know the deceased’s name and whom you will be speaking with. Use clear terms, like “dead” or “died” rather than euphemisms. Family members often want to view or sit with their deceased relative, and this should be encouraged, though not forced. (Note: occasionally, this may not be possible if the death is a criminal matter and

law enforcement has not finished its investigation of the body.) Prep family members ahead of time so that they know what to expect (are there visible injuries or medical interventions that may be disturbing?), provide a chair, allow them to touch or hold the deceased. If family wants a lock of hair, clothing, or jewelry, they should be allowed to take it (again assuming it does not interfere with law enforcement). If clothing was cut, provide an explanation of this to the family.

One intervention that is worth considering is to allow family to be present during resuscitation. There is a concern that it will be disturbing to family or that family will interfere with resuscitative efforts. Studies have shown that family do not compromise resuscitations and that witnessing the resuscitation typically has positive impacts on psychological outcomes for family. If this is to be implemented, a staff member should be delegated to remain with loved ones during the resuscitation.

Finally, death in the emergency department is not just stressful for family. Staff members can become distraught as well, especially during pediatric resuscitations, bad traumas, or even from knowing the deceased. Providing appropriate support services for staff is an important, though often overlooked, part of bereavement. Proactively engaging in appropriate treatment for family and staff affected by death is an important part of our role in the emergency department. We are going to have unsuccessful resuscitations. And when they occur, we need to remember that there are more patients than the one on the gurney.

## KEY POINTS

- Allow family to witness resuscitations when possible.
- Use a systematic approach for bereavement discussions.
- Use clear terminology; avoid medical jargon and euphemisms.
- Provide opportunities for viewing the body and for asking follow-up questions later (e.g., give family members your business card).
- Do not neglect care of staff involved in difficult/emotional resuscitations.

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# **DON'T BE AFRAID TO DISCUSS END-OF-LIFE DECISIONS WITH THE PATIENT AND FAMILY**

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**EMILY STREYER CARLISLE, MD, MA**

We see many patients near the end of their lives. By the nature of our training and our role in the health care system, the restoration of full health is our default goal. In many cases, however, a patient has come to the emergency department (ED) with a baseline so deteriorated or an injury so severe that full health is no longer a realistic or desirable goal, and the full deployment of our arsenal is inadvisable, if not futile. Initiating discussion about end-of-life care in such cases may spare the patient painful and pointless procedures, aid downstream decision-making, and conserve hospital resources.

The literature is unanimous that earlier discussion of end-of-life issues is better, especially for patients with terminal illness. Ideally, a primary care provider leads timely, unpressured discussions on end-of-life planning, and the transition from curative to palliative goals occurs in the patient's own time. Palliative care programs have been shown to reduce ED visits, hospital admissions, and aggressive care in the last month of life. In many cases, they improve symptom control and can even prolong life.

In other cases, however, these decisions are abruptly thrust at patients and their families by an unexpected or acute event. As emergency physicians (EPs), we often see these patients in the acute phase of the crisis. Giving bad news of any kind in the ED is challenging. We generally have no previously established relationship with the patient or family. The ED environment is often chaotic, loud, and anxiogenic. The urgency of the clinical encounter

often asks a patient or surrogate to make a high-stakes decision, with limited time for reflection, after receiving a bolus of new and often technical and at times, bewildering information. Nonetheless, it may fall to the EP to initiate these discussions. Furthermore, the ED encounter may be the last opportunity to determine the patient's wishes directly from the patient.

End-of-life discussions, as with all informed medical decision-making, require the physician to explain the options and provide guidance. The first step is to identify the patient and family's current understanding of the medical situation. They may not appreciate that an illness is terminal or that death is imminent, obvious as it may be to the provider. Take the family from old information to new, for example, "She has had trouble with her lungs for a while, but now her lung function is worse. Have you ever discussed what she would want in the event she could not breathe on her own?" Inquire about advanced directives or physician orders for life-sustaining treatment (POLST). At this point, discuss the options available in the ED and provide insight, for example, the more aggressive option might prolong life but is painful and imperiled with complications, whereas comfort measures prioritize peacefulness but likely fail to address the acute issue. Families might need help avoiding logically inconsistent preferences stemming from incomplete understanding (e.g., wanting vasopressors, understood as "medication to help blood pressure" but no central line).

As EPs, we routinely make life-and-death decisions in seconds, but for patients and families, these decisions may require time. Arriving at the decision to pursue comfort measures, especially when the event was unanticipated, may take multiple conversations over several hours to days. Patients and families may need time for adjustment, as well as the arrival of out-of-town relatives, or discussion with other physicians or advisors. Even if no decision is made, this first discussion in the ED may facilitate the later discussions with the inpatient team and allow a transition to palliative care that is both gentler and timelier. When time permits (e.g., need for intubation is not immediate), step away to allow time for reflection but remain available for questions. At this time, offer to call a palliative care team if available. The members of this team can offer answers and support to the family when the emergency nurses and providers often have so little time to spare.

Families may make the decision to pursue comfort measures starting in the ED. The time to death can be unpredictable and may be anywhere from minutes to hours to days. Unless death is obviously imminent (e.g., low oxygen saturations and bradycardia), admit the patient, preferably to a private, nonmonitored inpatient room, which allows family at the bedside while nature completes its course. While the patient waits for an inpatient bed, attempt to find a private room in a relatively quiet part of the ED. Call a

palliative care consultation if available and not already activated. Remember to address air hunger and pain to make patients as comfortable as possible.

## KEY POINTS

- Discuss end-of-life preferences whenever it seems appropriate.
- Be prepared to provide multiple small bundles of information, especially if the event leading to the ED was unanticipated.
- Call the palliative care team if one is available.
- If possible, try to move the patient and family to a quiet part of the department.
- Remember that the ED conversation will help the patient, family, and providers upstairs even if no decision is made in the ED.

## SUGGESTED READINGS

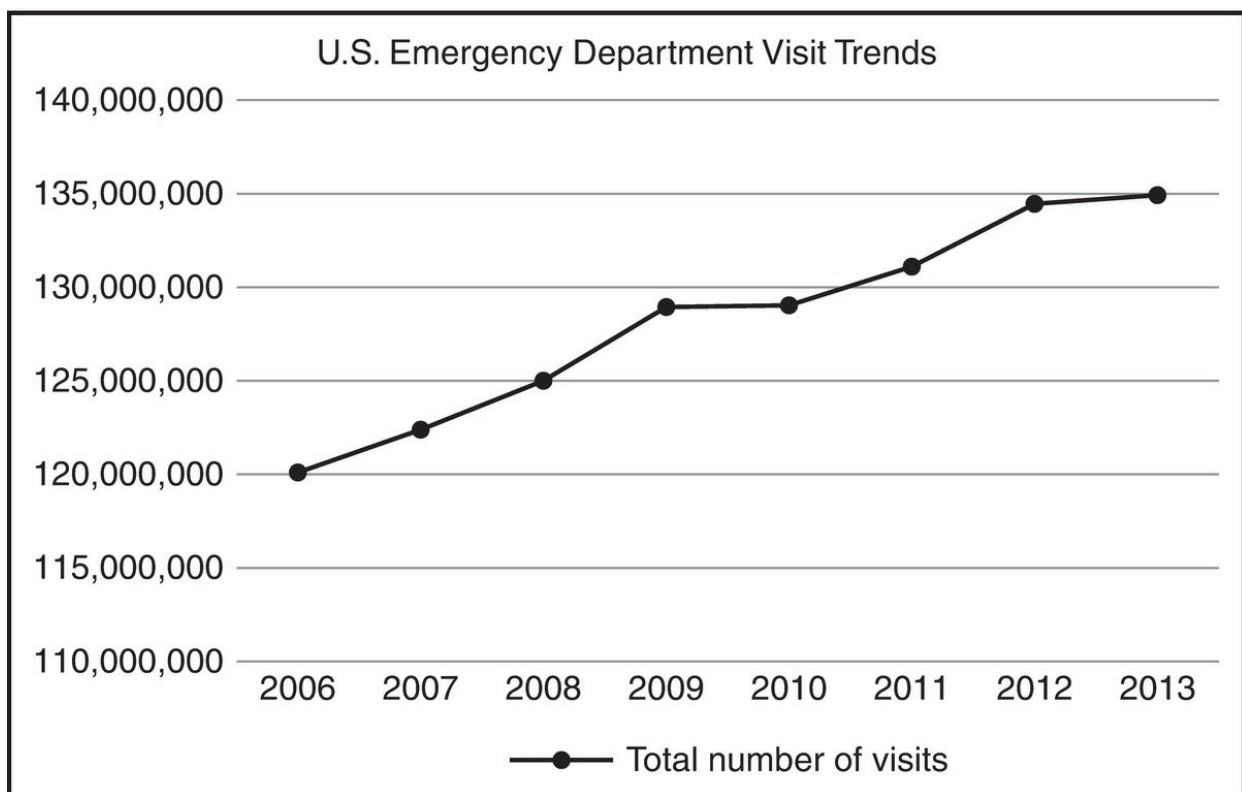
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# TOO MANY AT ONE TIME? EMERGENCY DEPARTMENT OVERCROWDING

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**RYAN BROOKS, MBA AND ARJUN CHANMUGAM, MD,  
MBA**

Emergency departments (EDs) across the country are finding themselves facing an increasingly more common situation—escalating demand of emergency resources and limited inpatient bed capacity, resulting in untenable overcrowding. For more than one decade, ED visit rates have steadily risen, contributing to overcrowding. ED overcrowding has a number of detrimental effects, including longer patient lengths of stay, increased morbidity, increases in adverse and preventable error, delays in treatment, and longer waits to be evaluated by caregivers. ED overcrowding leads to crowded waiting rooms and negatively impacts the patient experience, which also leads to decreased staff and provider morale ([Figure 359.1](#)).



**Figure 359.1** Emergency departments are tasked with caring for a steadily increasing number of patients.

## COMMON ERRORS IN OVERCROWDING

A number of strategies exist to combat the problem of ED overcrowding. Success of each of these strategies depends on the degree of leadership involvement across a health system. ED overcrowding is not a problem that is owned exclusively by emergency medicine. In fact, the ED component of overcrowding can be considered relatively small.

Recently, several publications have reported that the main cause of ED overcrowding is the extended boarding of inpatients in the ED. The lack of timely transfer of ED patients to appropriate inpatient beds is compounded by limited inpatient capacity and aggravated by hospital process inefficiency. When addressing this problem, leadership at all levels of the organization need to recognize that solving this problem requires coordination of the inpatient units, the ambulatory clinics, ancillary services, and even other hospitals within the health system.

It is helpful to consider two basic domains of variables that affect ED operations. The first domain includes the variables that are intrinsic to the ED itself. ED operations have to be optimized to reduce unnecessary waits, increase efficiency in throughput, and reduce dwell times for patients in beds. Bed turnover rates have a critical impact on the number of patients that

can be fully evaluated. Thus, improving bed turnover rates can help to optimize capacity and mitigate overcrowding. The second domain has to do with variables external to the ED, including efficiency of transfer of admitted or observation patients to other units, specialty consultation times, availability and accessibility of outpatient clinics, and ancillary services. The availability of outpatient referral services, including primary and specialty care clinic appointments, can have a significant impact on patients' ED dwell times. If clinic and outpatient resources are not readily available for referral by the ED physicians, the result is that prolonged evaluations and interventions will occur in the ED. This prolongs the ED evaluations, which exacerbate the crowding problem.

Unfortunately, many EDs are often faced to solve the problem of overcrowding in isolation of other hospital operations. A coordinated approach to overcrowding is the only way to achieve solutions. These solutions will be effective especially if there is integration of the ED services with other hospital-based services. A previous strategy had been to allow EDs to attempt to solve the problem independently of hospital operations, but such a strategy only address the initial element in the hospital-based health care continuum. Ignoring the other hospital services will only lead to longer ED patient waits, unsafe conditions, and suboptimal care.

Whatever strategy—or strategies—your organization decides to pursue, the most important thing to remember is that the problem will not get resolved without multidisciplinary intervention. Few EDs are resourced adequately to manage overcrowding in isolation of the other hospital services. Long-term ED planning must include an element of preparation for the potential of ever increasing demand for urgent and emergency care.

## KEY POINTS

- Over the last 10 years, ED visit rates have increased while inpatient bed availability has decreased, resulting in ED overcrowding.
- A leading cause of ED overcrowding is the boarding of admitted patients in the ED while they await inpatient beds.
- Other variables that affect overcrowding fall into two basic categories: factors that are intrinsic to ED operations and factors that have to do with hospital operations.
- Strategies to deal with ED overcrowding must be accomplished at the hospital operations level.
- Most EDs lack the required resources to solve the problems associated

with overcrowding, especially since overcrowding is often more a reflection of hospital operations rather than emergency operations.

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# DISCHARGE DOCUMENTATION: KEEP IT CLEAR, CONCISE, YET COMPLETE

DAVID ROSE, MD

Every patient leaves the emergency department (ED) eventually; either they are admitted or discharged. It is comforting when capable hands assume their care in the hospital, but often unsettling when they are sent into the world with only the advice we provide and a few pieces of paper.

Discharge instructions are a critical part of patient care and can alter the patient's course of illness. They provide essential information to the patient and to follow-up providers, as well as serving as a medicolegal document.

## AVOID COMMON ERRORS WHEN WRITING DISCHARGE INSTRUCTIONS

Include all essential elements of discharge instructions (see [Table 360.1](#)).<sup>1</sup>

**TABLE 360.1 ESSENTIAL ELEMENTS OF DISCHARGE INSTRUCTIONS**



|   |   |
|---|---|
| <b>Clinical Impression</b>                    | Give a diagnosis if known, or just an impression if you are unsure (e.g., don't call chest pain "gastroesophageal reflux disease," if unconfirmed)  |
| <b>Return Precautions and Expected Course</b> | Overall, the patient needs to know they should return if things are worsening or not getting better and should have an anticipated timeline for this. Specific recommendations can be helpful. For example, a patient with cellulitis discharged with antibiotics should return if erythema spreads or the patient becomes febrile. Giving nonspecific recommendations may prompt patients to return when it is not warranted clinically. For instance, telling a patient with chronic un concerning abdominal pain to return if they have abdominal pain can create confusion for the patient.                       |
| <b>Medications/Prescriptions</b>              | Review each prescribed medication with the patient, discuss what it is for, and state the most common and serious side effects. For example: "take oxycodone 1 tablet by mouth every 4–6 h as needed for severe pain not controlled by other over the counter medications; it may cause drowsiness or nausea."  |
| <b>Follow Up Information</b>                  | Be specific on follow up information including who, when, where and why. For example, "please follow up with you primary care doctor, Dr. Keefe, in 2–3 days at 5 West Hallow St, Baltimore, MD, 404-456-6847. Bring these instructions to follow up for your elevated blood pressure."   |
| <b>Aftercare Instructions</b>                 | If applicable provide aftercare instructions for things such as wound care and orthopedic injuries requiring splinting or casting.  |
| <b>Labs and Imaging</b>                       | Giving a patient a copy of their lab work and imaging reports can be very helpful for other providers and gives the patient an understanding of the workup completed during the visit. As advanced imaging becomes more prevalent, we are more likely to discover something unrelated to the patient's condition, but may require a more through investigation or follow up as an outpatient. As with all communication, being straightforward with these findings and ensuring appropriate and timely follow up is of great importance. Always document that you have communicated abnormal findings to the patient. |
| <b>Work and School Notes</b>                  | Knowing the occupation of your patient strengthens the doctor-patient relationship and allows individualized instruction on when it will be safe and appropriate to return to work. Bear in mind certain patients may require work restrictions based on their condition.   |

Adapted from Taylor DM, Cameron PA. Discharge instructions for emergency

## ***Use Preformatted Templates and Instruction Sheets***

Brief summaries of common conditions can help the provider construct discharge instructions more efficiently. These are commonly available via reputable medical sources online, or through your hospitals electronic medical record (EMR) system.

## ***Personalize Discharge Instructions***

The less generic and more personalized discharge instructions are, the more likely patients will actually comprehend and use them. Highlighting and going through the discharge instructions with the patient will aid in their understanding. Specific places to do this are in their “expected course” and “aftercare instructions.”

## ***Simplify Discharge Instructions***

When creating discharge instructions, “less is more.” The shorter and simpler the instructions are, the easier they are to follow. Furthermore, literacy in ED patients has been shown to average from a 3rd to 10th grade reading level.<sup>1</sup> This highlights the importance of the clear, simple language when communicating with patients, both verbally and in writing.

## ***Assuring Patients Demonstrate Understanding of Their Discharge Instructions***

ED patients frequently do not comprehend their discharge instructions.<sup>2</sup> Engel showed about 80% of patients lacked understanding regarding their home care instructions and return precautions.<sup>3</sup> Horwitz et al. found that with elderly patients who felt they understood their discharge instructions well, over half could not recall accurate information concerning follow-up appointments.<sup>4</sup> When discussing discharge instructions, the “repeat back method” or closed loop communication can be employed to assess comprehension, increase retention, and lead to better health care outcomes.<sup>5</sup> A brief discussion should complement the paperwork in order to make the discharge process more effective.

Next time you are writing discharge instructions, think of the time a patient came to your ED with a jumble of papers from an outside hospital

with essentially nothing useful on them. Write discharge instructions as if they were for one of your family members, as if you were the doctor at the next follow-up, or as if they were to be read to a courtroom.

## KEY POINTS

- Include all essential elements of discharge instructions.
- Preformed templates and instruction sheets help with efficiency and completeness of the discharge process.
- Personalize discharge instructions, and keep them simple in terms of language and content.
- Ensure patients demonstrate a clear understanding of their discharge instructions.

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## **RESIDENT AND ADVANCED PRACTICE PROVIDER SUPERVISION**

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**PATRICIA PETRELLA NOUHAN, MD, FACEP AND  
ROBERT B. TAKLA, MD, MBA, FACEP**

There is a balance between the ideal level of emergency medicine (EM) attending supervision and autonomy for EM residents as well as advanced practice providers (APPs; physician assistants and nurse practitioners). The use of APPs in the emergency center setting is commonplace but has been catalyzed by a number of factors including residency duty hour restrictions and the limited number of EM physicians in some settings. In addition, EM residency training programs require plans for graduated faculty supervision in order to ensure adequate teaching and patient safety. Rural emergency department (ED) settings may struggle to attract board-certified ED physicians and rely on APP employees to fill shift gaps. How much supervision of residents and APPs is needed, and what are the critical factors to keep in mind when doing so?

In EM residency training, there is a graduated progression of autonomy in practice from PGY1 to PGY2 and onward to more senior levels (PGY3, 4, or 5 depending on the EM program). The Accreditation Council for Graduate Medical Education (ACGME) requires PGY1 residents to receive direct face-to-face supervision or indirect with immediate availability of direct supervision at all times in the ED. As a resident progresses through their EM training, greater patient care responsibilities are assigned to them by their faculty and program director based on achieved skill level. They may advance to the level of seeing multiple patients before review and patient evaluation by the attending. Regardless of residency level, advanced procedures (such as intubation, chest tube insertion, or thoracotomy) should always be performed under direct faculty supervision. The ACGME

guidelines require supervision that is flexible based on resident level, resident skill, patient safety concerns, and available support services. The faculty must be attuned to these factors as they direct residents in safe patient care strategies and as they guide their learners to be future supervisors.

APPs do not have the same type of structured ED training as EM residents do, where they progress from year to year in their training specifically focused on EM. In addition, there is significant variability in state law dictating the independent practice of APPs. Depending on the APP school program, the student may be exposed to minimal EM. Instead, they gain ED knowledge, specialized skills, and experience on the job. Because of this variation, their level of required supervision and level of autonomy are very APP dependent. An NP who was an ED nurse for 12 years prior to completing her advanced training will require less supervision than one who is fresh out of school and has little ED experience.

For supervision of APPs, one suggested method is to define by protocol which types of encounters require that the patient be seen by the ED physician. Additionally, that encounter with the attending should be documented in the chart. For example, a repeat visit or high acuity patient may suggest the patient be evaluated by the ED physician and not discussed simply with a phone dialogue between the APP and the ED physician. These guidelines should be developed and agreed to in advance and tailored to state law, hospital bylaws, and departmental rules and regulations. A rural setting ED may only have an APP without any ED physician on site; therefore, the scope of practice and autonomy of that APP may be higher relative to an APP in an urban teaching hospital ED setting where there are EM residents and faculty.

What seems to be critical for optimal patient care and efficiency, as well as continued education and skill development of residents and APPs is honest open communication and feedback. If the APP or EM resident feels uncomfortable with any aspect of patient care, they need to ask for assistance and supervision without hesitation. Anything less jeopardizes patient care as well as the integrity of the educational process for both types of learners.

## KEY POINTS

- There is a balance between the individualized autonomy given to each EM resident and APP based on their skill set and experience coupled with the supervising physician's level of comfort with that particular learner.

- PGY1 EM residents require direct supervision by EM faculty as dictated by ACGME standards.
- Adequate senior EM resident supervision allows more flexibility for the faculty oversight yet critical patient evaluation and procedures still require direct face-to-face supervision.
- The autonomy and scope of practice appears greater in rural ED settings for APPs due to the lack of EM board-certified physicians.
- Open communication is essential between attending physician and resident or APP for effective and safe patient care and supervision.

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# WHAT TO DO WITH SO MANY? STRATEGIES FOR REDUCING EMERGENCY DEPARTMENT OVERCROWDING

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**RYAN BROOKS, MBA AND ARJUN CHANMUGAM, MD,  
MBA**

Possessing the ability to anticipate emergency department (ED) overcrowding is one of the most valuable and cost-effective strategies an organization can employ. Historical demand for emergency services can be analyzed to provide a sense for when to expect peak patient arrivals. Tracking arrival patterns by day of the week and hour of the day allows leadership to adjust staffing patterns to better match the demand for their services. Arrival patterns are the most basic element of EDs that needs to be understood. Secondary to arrival patterns are a better understanding of the occupancy levels on inpatient and observation units. Ideally, the highest arrival rates would match closely with when the hospital has the lowest occupancy. Matching the supply of available inpatient beds with the demand for these beds is the ultimate objective.

An example shown below uses conditional formatting to easily identify when ED census is greatest. Using the data below, nursing directors may decide to shift an additional RN into an afternoon shift as opposed to a lower census morning shift. The data below also show when available inpatient and observation beds are most needed. As arrival rates rise, having the ability to offload patients from the ED is key to success. Having patients wait in an overutilized ED while a bed becomes available only exacerbates the problem

of ED overcrowding, impacts the patient experience, and can potentially complicate patient and provider safety. Morbidity and mortality is adversely affected when an ED patient is forced to wait hours for an appropriate intensive care unit bed. Unfortunately, this scenario is becoming all too common in EDs across the United States ([Figure 362.1](#)).



| Average ED Census - Jul - Nov FY16 |      |      |      |      |      |      |      |
|------------------------------------|------|------|------|------|------|------|------|
| Adult Patients Only                |      |      |      |      |      |      |      |
| Arrival Hour                       | Sun  | Mon  | Tue  | Wed  | Thu  | Fri  | Sat  |
| mdnt – 1a                          | 42.1 | 41.4 | 48.3 | 43.0 | 38.2 | 46.9 | 39.0 |
| 1a–2a                              | 40.1 | 43.3 | 39.9 | 37.3 | 35.1 | 43.0 | 31.3 |
| 2a–3a                              | 39.3 | 36.1 | 33.8 | 31.8 | 36.9 | 40.6 | 34.3 |
| 3a–4a                              | 41.7 | 35.4 | 30.3 | 28.9 | 33.8 | 35.6 | 32.3 |
| 4a–5a                              | 33.6 | 30.8 | 25.0 | 30.0 | 35.1 | 32.7 | 31.6 |
| 5a–6a                              | 30.3 | 25.6 | 27.0 | 28.1 | 34.7 | 24.2 | 31.5 |
| 6a–7a                              | 31.8 | 28.1 | 25.2 | 22.2 | 23.9 | 24.2 | 24.9 |
| 7a–8a                              | 28.3 | 27.1 | 25.3 | 24.4 | 23.6 | 22.3 | 25.3 |
| 8a–9a                              | 24.9 | 23.6 | 26.2 | 29.3 | 26.5 | 24.6 | 26.9 |
| 9a–10a                             | 23.5 | 31.1 | 27.2 | 31.9 | 29.6 | 25.3 | 26.4 |
| 10a–11a                            | 29.7 | 39.8 | 34.9 | 39.1 | 34.1 | 30.8 | 30.3 |
| 11a–noon                           | 32.9 | 47.9 | 41.8 | 43.4 | 41.0 | 38.4 | 29.1 |
| noon–1p                            | 36.1 | 54.1 | 45.5 | 50.5 | 46.3 | 41.4 | 36.0 |
| 1p–2p                              | 35.4 | 59.6 | 48.2 | 55.7 | 50.9 | 44.5 | 38.9 |
| 2p–3p                              | 39.9 | 62.0 | 50.7 | 56.4 | 49.8 | 46.3 | 42.0 |
| 3p–4p                              | 35.6 | 62.5 | 51.8 | 58.2 | 52.8 | 48.0 | 44.1 |
| 4p–5p                              | 41.2 | 65.1 | 56.9 | 57.6 | 58.1 | 53.1 | 48.9 |
| 5p–6p                              | 37.7 | 68.0 | 58.6 | 54.6 | 56.6 | 53.4 | 43.3 |
| 6p–7p                              | 41.2 | 64.2 | 62.5 | 55.8 | 56.3 | 52.9 | 40.2 |
| 7p–8p                              | 43.4 | 60.8 | 52.6 | 53.5 | 55.9 | 42.3 | 40.3 |
| 8p–9p                              | 47.1 | 56.4 | 57.8 | 56.9 | 48.8 | 44.2 | 40.6 |
| 9p–10p                             | 48.7 | 58.6 | 58.0 | 55.1 | 52.8 | 49.7 | 34.9 |
| 10p–11p                            | 48.9 | 52.2 | 56.3 | 50.9 | 50.2 | 45.4 | 41.1 |
| 11p–mdnt                           | 40.0 | 49.0 | 45.1 | 42.1 | 52.4 | 43.8 | 42.3 |

**Figure 362.1** Historical ED census data should be analyzed to align staffing with demand for services. When possible, creating inpatient capacity when the ED census is highest will improve throughput times and patient safety.

EDs cannot wait until overcrowding has become a problem before it is addressed at a hospital-wide level. Advanced planning is a must when strategizing against overcrowding. Metrics that can determine when planning must begin can be set by each organization. The most commonly used are volume metrics including ED census and ED boarding rates (number of patients awaiting an inpatient bed but remaining in the ED) and time-based

metrics such as how long it takes for patients to be evaluated by a provider in the ED or how long it takes for a patient to get a room in the ED. Predetermined actions can be taken based on an ED census of that is patients is 40% greater than the number of beds or when the time to provider metric exceeds 90 minutes.

One of the most common factors leading to overcrowding is lack of inpatient bed availability. By creating discharges earlier in the day, hospitals may be better able to match the demand for inpatient beds (ED and OR patients awaiting admission) with the supply of open and staffed beds. To encourage such behavior, many hospitals are implementing daily fixed discharge targets for each inpatient unit. An example of this strategy is instructing inpatient units to discharge two patients each day by 10:00 am. By doing so, the ED then has inpatient bed availability that reduces boarding patients and ultimately creates room for incoming ED arrivals. To accomplish this, a dedicated discharge team can help to enact safe, appropriate discharges, ensuring that patients will do well in the ambulatory setting. Some institutions are developing a new specialty clinic that will see patients who were recently discharged from the hospital as they transition to more traditional longitudinal care. Developing such transition clinics have two functions: (1) ensure that inpatients have an appropriate discharge care plan in an effort to efficiently transition them to ambulatory care and thereby reduce hospital length of stay and (2) provide ED patients with options for outpatient follow-up, including the multidisciplinary transition clinic.

Discharge lounges are often created to make it easier for inpatient units to discharge patients earlier in the day. These lounges can be utilized by patients who are otherwise ready to go home but are unable due to transportation or other minor obstacles. Allowing these types of patients to stay in a lounge area and not in a needed bed can be key to reducing ED crowding. The most common barrier reason given for not creating a discharge lounge is the impact to patient satisfaction. Yet few studies exist to demonstrate the impact of well constructed, dedicated discharge lounges outfitted with the appropriate services on the patient experience. Sadly many hospitals neglect the serious impact of boarding instead of focusing on how to better utilize inpatient beds especially with patients whose medical interventions are complete and are only waiting for discharge.

If mandating discharges is unfeasible—or unpopular—in an organization, an alternate strategy is to create a multidisciplinary (including both clinical and nonclinical) approach to capacity planning. Morning bed meetings can be scheduled each morning to plan for the day. Emergency department managers and charge nurses connect with inpatient unit charge nurses to determine where the greatest bed needs are. By starting with the

current census on each unit and identifying how many patients can be expected during the day (based on estimated historical data and what the ED and OR is currently holding), hospitals can easily identify where beds are most needed. These meetings should not just be focused on sharing information, which is the first step. Developing a clear daily action plan with accountability and shared responsibility for resolving issues is critical, but often not done.

For example, a 30-bed unit with 28 current patients and 4 anticipated admissions knows that to accommodate the needs of the ED requires two patients to be discharged. Having all of the needed participants allows for units to be specifically targeted for discharge help. Transport teams know that units with high demand will be the priority. Pharmacy teams can begin planning take-home medications for these patients. The key to success using this strategy is eliminating as many barriers to discharge as possible. If it is simply not feasible for discharges to be made to accommodate the needs—other units are asked to step up and take these admissions if possible.

This strategy—aligning demand and capacity in real time—can be extremely successful at a hospital level. However, this strategy can also be taken and applied at a health system level as well (particularly when hospitals within a health system are located in close proximity to each other). If two hospitals within the same health system are serving the same area, it is wise to understand in real time the available capacity and demand from each of the respective hospitals. Many consider the ideal occupancy rate of hospitals to be 85%. When hospital capacity reaches 85%, it becomes increasingly more difficult to place patients in appropriate units, make patient transfers to appropriate units and to cohort genders and certain isolation patients.

When one of these hospitals surpasses the ideal occupancy rate (85%), the sister organization should consider transferring ED boarding patients (awaiting an inpatient bed) to the other hospital. Creating an additional step of transferring the patient is a much smaller obstacle to overcome than trying to create capacity by discharging patients earlier. If patient satisfaction is a concern, patients can even be given the option to transfer if they would like. A short ambulance transfer could put them in an inpatient bed much sooner than if they choose to stay at a full hospital awaiting a bed. Many times the patient will even appreciate the choice being offered to them—resulting in greater patient satisfaction/experience scores regardless of what decision the patient makes.

Another popular option to address ED overcrowding has been to create or leverage access of preexisting clinics. Often times, organizations who are

trying to reduce their ED overcrowding are at the same time trying to increase the visit rates to their outpatient clinics. Outpatient clinics are a great resource to many ED patients—many of whom could be seen in an outpatient setting without worry. The key to making this strategy as successful as possible is identifying the types of patients who would benefit from such an offering as early as possible during their ED visit. Equally important is to ensure that there is appropriate access and a means to facilitate patient engagement with these clinics.

Following a medical screening exam, if it is determined that a patient emergency does not exist, patients can be offered clinic appointments as an alternative to ED care, but these appointments need to be 24 hours to 2 weeks depending on the patient condition and preference. Once patients have been evaluated and informed that they are safe to wait for care for another 1 to 2 days, they often appreciate having a scheduled appointment that conforms to their schedule and preferences.

Remember, ED visits are usually unplanned and stressful. Whenever possible, offer patients a better experience that will also help to reduce overcrowding.

ED overcrowding is a nationwide problem, and many forecast that ED visits will only continue to rise. Deploying specific strategies at both a hospital level and a health system level can greatly affect ED crowding, staff morale, patient safety, and the patient experience.

## KEY POINTS

- Developing mechanisms, such as tracking arrival patterns to predict ED census is a valuable strategy in managing potential ED overcrowding situations.
- Volume metrics including ED census and ED boarding rates (number of patients awaiting an inpatient bed but remaining in the ED) and time-based metrics such as how long it takes for patients to be evaluated by a provider in the ED or how long it takes for a patient to get a room in the ED can provide useful data to help with planning for high census conditions.
- Other strategies, including transferring to other hospitals, encouraging more efficient inpatient discharge mechanisms, and utilizing outpatient clinics more effectively are some strategies to improve hospital capacity.

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## **WHAT TO DO WHEN THE REGISTERED LETTER ARRIVES**

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**KEVIN M. KLAUER, DO, EJD, FACEP**

Fear of the unknown is often the initial response of physicians when they receive a notice of intent to sue. With respect to medical malpractice, physicians are often like ostriches with their heads in the sand. There is such an aversion to the legal process, that many physicians would prefer to avoid the claims process altogether rather than prepare themselves by understanding the process. This is a failed strategy. What you don't know can definitely hurt you. The best approach is being prepared. Sadly, a common sense approach to understanding the complexities of the legal system is simply not good enough because the process is not always logical. Many physicians will get sued; understanding and employing the strategies outlined in this chapter can help to better manage a very challenging and stressful time.

The initial notification of a claim will likely come as a certified letter and often times as a notice of intent to sue or a demand letter. Such notices should outline the parties, the claim(s), and allegations, and if there is a settlement demand, will include a summary of damages and the proposed monetary settlement amount. However, your first notification may be that of the claim and lawsuit, following its filing with the court.

There are three distinct areas to focus on, regarding receipt of a notice of intent to sue, demand letter, or notification of being named as defendant in a lawsuit: Timely response, privileged communication, and your health.

Although states vary in their rules and approach to civil procedure and adjudicating allegations under the tort of negligence, there are guiding principles that are broadly applicable and often useful. A response to the

notice should be prompt. Understand that if the defendant is served with notice of a lawsuit, a lack of timely response may result in summary judgment for the plaintiff. There are too many horror stories of physicians who have buried their head in the sand by filing away their notice in a desk drawer. The physician defendant should notify their supervisor, insurance carrier, and risk/claims management department immediately. Upon notice, these entities should initiate development of a case file, a defense strategy, and a proper legal response. It is never appropriate to respond on your own behalf without legal advice. By all means, do not reach out to the plaintiff (your former patient) without consultation and participation of your defense counsel.

It is a common reaction for providers to want to discuss the details of the case with colleagues to garner support and to validate their competency. However, this can be risky. One of the first questions asked in a deposition will be if you have discussed the case with anyone and if so, with whom? Discussions with colleagues are usually not privileged. Although some conversations may fall under a given state's peer review protections, such protections are variable and the extent of privilege is often vastly overestimated. The strongest protection of privilege is the attorney-client privilege. However, the protection afforded under this privilege is frequently misunderstood. In short, these communications are protected by the privilege, cannot be divulged by the attorney and are not discoverable. Yet, the communication that is protected is only that which extends from legal representation. Thus, talking to an attorney who is your family friend is not privileged unless they are representing you in that specific matter. Simply typing "attorney-client privilege" in an e-mail subject line does not necessarily afford you protection. For instance, when you have copied others on that e-mail that are not subject to that protection, a waiver of privilege may occur.

Spousal privilege and clergy-penitent (e.g., bona fide clergy at the discretion of the court's determination) privilege do afford strong protection and opportunity to safely discuss your case. Again, such communications may be inadvertently waived if those parties speak to others or others who are not subject to the privilege are included or overhear the disclosure(s).

Medical malpractice is simply daily business and frequently sport for the plaintiff's attorney. However, to physicians and other medical providers, this is personal, which may lead to significant stress and mental health issues. It is important to recognize that this process masquerades behind a cloak of truth and justice. However, the attorney's role is to gather and understand the facts of the case and to support their client's position to the best of their ability. Their objectives do not include an imperative to find absolute truth in

the matter.

Physician suicide is much greater than that of the general population, 40% greater in males and 130% greater in females, with medical malpractice, litigation being one of the most common triggers. Litigation stress support is critical. It is not a question that litigation causes stress. It's a matter of the extent of stress that it causes and how it will impact the individual defendant(s). Support should be sought via mental health professionals, legal counsel, spouse, and clergy, and such support should be ongoing. Waiting for overt signs of depression, substance abuse, or dependence and other forms of decompensation will only result in treatment delays and may result in suicide. Although physicians are taught to be strong and function in isolation, no one should be expected to navigate these uncharted and unfriendly waters alone.

## KEY POINTS

- Make certain you report the notice to the appropriate persons immediately upon receipt.
- Establish defense counsel early to make certain you have representation and preserve your rights and understand the protections afforded to you.
- Confide in those with whom you can have privileged communications with.
- Don't let the filing of one case change how you care for thousands. Bad outcomes do not always result from bad care.
- Seek out litigation stress support. It is not a sign of weakness.

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## **YOUR DEPOSITION**

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What we do not understand will not only be anxiety provoking, but can result in self-victimization from the “law” of unintended consequences. Your deposition is a prime example. A deposition is defined by Black’s Law Dictionary as, “A witnesses out of court testimony that is reduced to writing for later use in court or for discovery purposes.”

What is conspicuously absent from that definition is, “This is the doctor’s day in court.” This is, perhaps, the greatest and most dangerous misunderstanding that physicians have of the legal process.

Your deposition will be scheduled early in the course of a medical legal case, following filing of the lawsuit. The deposition is the cornerstone of the discovery phase. Many decisions will be made based on your deposition performance, which may even include dismissing the claim(s) against you. However, if the plaintiff’s attorney has done their homework, dismissal is very unlikely, as the goal of the deposition isn’t to “find facts,” but rather to find facts to support specific arguments and theories of allegation.

Generally speaking, there are several goals of a deposition: Testing existing theories/allegations, identifying new theories, sizing you up, and setting the trap.

Prior to ever considering pursuit of a case, the plaintiff’s attorney will have secured experts to review the case and provide an opinion regarding whether or not the care fell below the standard of care and what aspects of the care were substandard and are defensible or indefensible. These experts will provide the basis for the claim and the allegations against you. Without an expert to support their claim(s), there is no way to substantiate the claim, and thus, no lawsuit. However, these theories and allegations must be tested. Does the defendant have a plausible explanation? Are there additional facts

that would undermine their theories? These can be mined for through the deposition process, yours and others, including experts who may be called to later testify at trial.

To a large extent, depositions are fishing expeditions. Such fishing expeditions may yield new facts, new insights, and even new theories/allegations and avenues to pursue in the lawsuit. That is why, when being deposed by a plaintiff's attorney, it is your day to be as quiet as possible and not "your day in court." Make them do the work. Do not fall victim to the well-intentioned urge to explain yourself in the hopes of convincing them how wrong their allegations are. The more you say, the more is transcribed, and the more you will be accountable for at a future date, in particular, at trial. In other words, if they want to fish for facts to use against you, don't throw the fish in their boat. Make them work for it.

Much of the discovery phase of a lawsuit is determining strategy. Another very important aspect to plaintiff and defense strategies is their determination of what kind of witness you will make. Are you credible? Will the jury like you? Are you overtly nervous while providing testimony? Can you defend your actions and the care you provided? These, and many more, questions will be answered from your deposition. As a matter of fact, they are likely to audio or video your deposition for this exact purpose. If you are a good witness, this will play well for you. If you aren't, this is more opportunity and leverage for the plaintiff's attorney to pursue higher settlement demands. They may use this opportunity to rattle their sabers and intimidate the defense team, proclaiming their chances of a plaintiff verdict at trial are greater now that they have sized you up.

Perhaps, the most challenging part of the deposition is maintaining consistency of your message. This can be mentally taxing. The last thing you want is the plaintiff's attorney using your own words to discredit you, strengthening their case. It is truly bad enough that they can and will secure expert witnesses to weaken your case and strengthen theirs, but it is devastating when the defendant aids them in this crusade. As previously noted, the plaintiff's attorney has theories, and they intend to prove them and prove them at your expense. They are likely to ask you the same exact question or similar questions three, four, or more times. It can be maddening, but don't lose your focus. This is a game of cat and mouse. They are trying to shape your testimony in a way that they would like and can use, and they will attempt to set the trap.

The trap is a game of mental confusion. If they ask you a question enough times or in enough different ways, you are likely to contradict yourself. Make no mistake. These missteps, realized by you and your defense

counsel or not, will come back to bite you. The defendant will be asked a similar or identical question at trial, and such self-incriminating statements will be recited in front of the jury. The plaintiff's attorney may even ask you to read that portion of your deposition testimony. Of course, at that point, no one will know what trickery occurred to extract that particular response from you. It will simply appear that on the day of your deposition you said one thing, and in the courtroom, you said something else. Such contradictions are at least undermining of a defendant's credibility, if not devastating to their case. So, be smart, be consistent, and be quiet. The least said in a deposition, the better off you are.

Preparation for your deposition is critical. Mock depositions to prepare for anticipated lines of questioning are essential. Your defense counsel can assist with helping to craft your messages and get you comfortable with the process. Plan on a long day. You should be well rested and well fed. Although it seems like a fine line, you're being deposed, not interrogated. If you need a break or want a break for any reason, just ask for one. Dress in business attire, be respectful, be truthful, and maintain good eye contact. If you don't understand a question, don't answer it until it is clarified.

## KEY POINTS

- Understanding the purpose of the deposition is for fact finding, but not to discover the truth.
- Consistency of well thought out responses to difficult questions is critical to protecting yourself.
- Approach this like an important exam: Study, get plenty of rest, and eat a good meal in advance.
- If you don't remember, simply respond, "I don't remember."
- If at all possible, be cordial, positive, engaging, and maintain eye contact.

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## SURVIVING A LAWSUIT

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**HUGH F. HILL III, MD, JD, FACEP, FCLM**

Your patient's bad outcome was painful, but now you are accused of causing it! Or what you thought was a success is now called a failure, in a claim that states you should have done even better. Or, perhaps least painful but most infuriating, nothing bad happened and yet you are still accused of error.

We understand intellectually that people harmed by wrong actions or failure to act should not have to bear the costs, that the individual or institution responsible should compensate the innocent victim. We may accept the economic argument that the person best able to potentially avoid the damage should be accountable. We may recognize that the "system" doesn't care so much about fairness; society simply prefers that people take their disputes to court rather than fall back on feud, vendetta, and violent alternatives.

Every rationale is cold comfort when a health care provider is served their subpoena in a medical malpractice lawsuit. For most health professionals, compensation is only part of our motivation. We do what we do to help patients; to be accused of causing harm assaults our *raison d'être*. It's the rare provider who can take being sued as "just business." Some describe the experience as comparable to the death of a close friend or relative.

If a law suit were to occur, we know that it will involve a very public humiliation, and we will be nearly helpless as our knowledge, awareness, and choices during a busy shift are dissected and criticized. Jurors without any understanding of medicine, and likely little of science, are going to decide whether or not we are at fault.

And the aftermath: Every application for licensure and privileges asks, "Have you been sued." Now the answer will be yes and require explanation.

Any award or settlement will be reported to the national data bank. Even if you win, you are no longer a cost-free doctor to the insurers. Professionals report a sense of shame about being sued, even when convinced their care was correct. Posttraumatic stress symptoms appear. And significantly, a whole new source of potential bias in your practice must be monitored, for example, if the case involved an assertion that you should have CT'd the plaintiff, will you now over-radiate patients?

Coping strategies exist. Some are obvious and natural, some may require overcoming psychological barriers. There are also important “don’ts”; it is possible to make the situation worse.

## Do

- **Acknowledge your feelings.** Anger and depression are universal responses. Compartmentalization comes at a cost. We believe the physicians who have killed themselves after being sued already had problems, but it’s hard to know. Denial is not the ideal coping strategy.
- **Share.** This is difficult. The sense of shame and fear of being judged block our impulses to seek feedback and affirmation. Our significant others need to know what’s going on. We need to hear repeatedly what we already know: no matter what the plaintiffs and their lawyers say about us, we are good people and caring professionals. Even if you are limited to talking about feelings, it’s still helpful. NOTE: You may be asked under oath if you have discussed the case with anyone else. Your attorney will know the law in your jurisdiction and with whom and what you can share.
- **Consider recording your current recollections about the case.** Date it properly. Litigation can take years before it actually comes to trial. The plaintiff is one of many patients or families you have cared for, whereas the plaintiff will have a rock solid memory of what happened, reinforced over time.
- **Regain a sense of control.** Try to overcome that wish to deny and take a very active role in the defense, at every stage. Research literature addressing the care being criticized, be aware of when it was published. Meet with insurance carrier–assigned counsel early and often. They will often say they are working for you, but they may have other allegiances. Both the lawyers and you should be aware of potential conflicts of interest. Consider hiring your own lawyer out of your own pocket as additional quality control and support. The expert witnesses will be crucial. You should be involved in your counsel’s choice of expert by researching their publications and history; you can be sure the other side

will. And when you know the plaintiff's experts, dig in. All their publications and presentations should be exhumed and examined. Although it's not often done, we recommend you try to attend as many of the depositions of the experts as possible. If you miss one, read the transcript especially carefully. Their demeanor at deposition will suggest what it will be at trial. If the same lawyer that's suing you deposes and questions at trial, you'll be more prepared.

- **Professional counseling is available.** Talk therapy and behavioral therapists stress mitigation techniques can help. ACEP has offered, and may still at the time you are reading this, peer-to-peer counseling for defendant emergency physicians through the Medical Legal and Wellness Committees.
- **Talk with your counsel about possibly issuing a demand letter.** Consider telling your insurance carrier that you want them to offer policy limits. If they refuse and the court awards more money than the policy covers, you may then have a cause of action for the additional amount against the insurer. This is especially important if the plaintiffs are demanding more than policy limits. Prepare to Testify. Before being deposed, your lawyer will want to prepare you. Insist on as much time as you need. Generally, you will be advised to answer only the question asked and refrain from volunteering information. Most plaintiff's counsel try to encourage you talk in deposition; they save the attacks for court. If you or your lawyer are concerned about your appearance in court, there are services that consult on this, and even those that will organize a mock trial of your case before the real thing.

## Do Not

- Don't attempt to alter or destroy records, even if they are incorrect or misleading. Defensible cases have been rendered losers by revelation of backdated, altered, or lost records.
- Don't speak directly with plaintiffs, their experts, or their experts' colleagues. This could be interpreted as harassment. And don't talk with anybody on the other side, or any strangers, without your lawyer present. Don't discuss the facts of the case with colleagues or other defendants unless your counsel approves. You will feel the need for absolution by peers, but the discussion could become an element of the case, or your friend could be drug in as a witness.
- Don't misrepresent facts to your counsel. Yes, you want her deeply committed to your interpretation of what happened and you are advocating for that interpretation, but she will be hobbled by any



misunderstanding.

One last bit of advice: Remember you will get through this. It will take about a year after the case is over before things start to feel normal again. You may have to go through every stage of grief, but you will return to what makes our work the joy that it is, caring for and about patients you serve.

## KEY POINTS

- Acknowledge your feelings
- Share and seek help
- Take back some control by active involvement in the process
- Don't mess with the records
- Never lose faith that you will survive this

## SUGGESTED READINGS

- Andrew L. GPS for malpractice litigation. *Emergency Physicians Monthly*. Available at: <http://epmonthly.com/article/gps-for-malpractice-litigation/>
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- enhancement of elimination

- indications

- severe metabolic acidosis

- sodium channel blockade

STEMIs

surgical airway

- contraindications

- post cricothyrotomy

- preparation

- procedure

tension pneumothorax

- “the pipes”

- “the pump”

- “the tank”

therapeutic hypothermia

- American Heart Association (AHA) guidelines

- CPC/ modified Rankin scale score

- HACA study group

- out-of-hospital cardiac arrest

- targeted temperature management

vascular catastrophes

- AAA rupture

- aortic dissection

vasopressors

- class of drugs

- selection of therapy

- shock
- Surviving Sepsis guidelines
- vascular access
- vasoactive agents
- venous thromboembolism (VTE)
- C-reactive protein (CRP)
- Cricoid pressure (CP)
- Cricothyrotomy
  - caustic ingestions
  - contraindications
  - post cricothyrotomy
  - preparation
  - procedure
- Critical care
  - abdominal compartment syndrome (ACS)
  - ACS
    - management algorithm
    - primary
    - recognition and diagnosis
    - recurrent
    - risk factors
    - secondary
    - trans–bladder pressure measurements
  - benzodiazepines
  - deep vein thrombosis (DVT)
  - end-of-life (EOL) care in
  - excess fluid administration
  - extracorporeal life support (ECLS)
  - fluid resuscitation
    - arterial waveform analysis
    - end-tidal carbon dioxide
    - inferior vena cava, ultrasound assessment
    - passive leg raise
  - intra-abdominal hypertension (IAH)
    - management algorithm
    - recognition and diagnosis
    - risk factors
    - trans–bladder pressure measurements

- nonsteroidal anti-inflammatory medications (NSAIDs)
- novel oral anticoagulant (NOAC) medication
- oversedation and undertreatment of pain
- plateau pressure
- red blood cell transfusion
- traditional vitamin K antagonist (VKA)
- unnecessary blood transfusion
- ventilator-associated pneumonia (VAP)
- Crohn disease (CD)
- Croup
- Crying
  - colic, diagnosis of
  - IT CRIESS mnemonic
  - life-threatening conditions, primary/sole manifestation
  - systematic approach
- Crystalloids
  - bolus
  - cold
  - fluids
  - hydroxyethyl starch (HES)
  - infusion
- CSF *See (Cerebrospinal fluid)*
- CTA *See (Computed tomography angiogram)*
- Cutaneous
  - cellulitis
  - chickenpox and shingles
  - erythema nodosum, nodules and hypersensitivity
  - necrotizing soft tissue infection (NTSI)
  - rashes
    - classic pediatric rashes
    - classic tick-borne illnesses
  - Stevens-Johnson syndrome (SJS)
    - barrier, loss of
    - disposition
    - internal involvement
    - skin findings
  - toxic epidermal necrolysis (TEN)
    - barrier, loss of

- disposition
- internal involvement
- recognition of
- SCORTEN system
- skin findings
- treatment guidelines for

CVST *See (Cerebral venous sinus thrombosis)*

Cyanide toxicity

- cause of
- clinical manifestations of
- partial pressure of oxygen
- systemic toxins

Cyanotic postoperative pediatric cardiac patients

- bidirectional Glenn
- Hemi-Fontan
- hypoplastic left heart syndrome
- overcirculation
- pulse oxygen
- repair

## **D**

Damage control *See (Balanced resuscitation)*

D-dimer assay

DeBakey classification

Decompression sickness (DCS)

- type I
- type II

Deep space neck infection

Deep vein thrombosis (DVT)

Deferoxamine

Delayed sequence intubation

Delirium

- diagnosis
- etiologies of
- geriatrics
- Thomas A. Swift Electric Rifle
- treatment

Delirium tremens (DTs)

- Dementia
- Depression
  - evaluation of
  - medical conditions
  - postpartum
  - PR-segment
  - symptoms of
- Dermal (deep) sutures
- Dexmedetomidine
- Diabetic ketoacidosis (DKA)
  - hyperglycemic hyperosmolar state (HHS)
  - insulin
  - intravenous fluids
  - potassium
  - sodium bicarbonate
- Dialysis access
- Dialysis disequilibrium syndrome (DDS)
- Diarrhea
  - appendicitis
  - gastrointestinal, mycophenolate
  - hypomagnesemia
  - plasma bicarbonate concentration
  - sigmoid volvulus
  - traditional mths
- Diazepam (Valium)
- Diffuse interstitial lung disease
- Digital subtraction angiography (DSA)
- Digoxin
  - atrial fibrillation
    - rhythm control
    - and RVR
  - cyanide
  - toxicity
- Dilemma
- Diltiazem
- Direct thrombin inhibitors (DTIs)
- Distributive shock
- Dizziness

- acute coronary syndromes
- acute mountain sickness (AMS)
- anemia
- carbon monoxide toxicity
- causes of
- dialysis disequilibrium syndrome (DDS)
- head CT
- history
- physical exam
- symptom of
- treatment
- vertigo
- DKA *See (Diabetic ketoacidosis)*
- Dobutamine
- Double bubble sign
- Dramatic disorders
- Drug-seeking behavior
- DTIs *See (Direct thrombin inhibitors)*
- DVT *See (Deep vein thrombosis)*
- Dynamic markers
- Dysrhythmia
  - ECG artifact from
  - electrical injuries
  - multifocal atrial tachycardia
  - oxygen desaturation
  - ventricular assist device (VAD)
  - wide complex tachycardia (WCT)

## **E**

### **Ear**

- auditory abnormalities
- injury
- lacerations
- malignant otitis externa (MOE)
- tympanic membrane
- EASI *See (Elder Abuse Suspicion Index)*
- Ebola virus disease (EVD)
- ECG *See (Electrocardiogram)*

- Echocardiography
- Eclampsia
- ECLS *See (Extracorporeal life support)*
- ECMO *See (Extracorporeal membrane oxygenation)*
- Elder abuse
- Elder Abuse Suspicion Index (EASI)
- Electrical burns
- Electrical injuries
  - associated injuries
  - contact time
  - forms
  - household/industrial
  - pathway
- Electrocardiogram (ECG)
  - aortic dissection in
  - atrial fibrillation (Afib)
    - multifocal atrial tachycardia (MAT) and
    - with rapid ventricular response (RVR)
    - rate control vs. rhythm conversion
    - in Wolff-Parkinson-White syndrome
  - chest pain evaluation
  - dysrhythmia
  - pediatrics
  - syncope
    - arrhythmogenic right ventricular dysplasia (ARVD)
    - Brugada syndrome
    - catecholaminergic polymorphic ventricular tachycardia (CPVT)
    - hypertrophic cardiomyopathy (HOCM)
    - prolonged QT syndrome
    - short QT syndrome
    - ventricular preexcitation
  - VT and SVT
- Emergency medical services (EMS)
- EN *See (Erythema nodosum)*
- Encephalitis
- Endocrine/metabolic physiology
  - acid-base disturbances
  - acute respiratory acidosis

- acute respiratory alkalosis
- anion gap
- bicarbonate concentration ( $\text{HCO}_3$ )
- bicarbonate therapy
- chronic respiratory acidosis
- chronic respiratory alkalosis
- diabetic ketoacidosis (DKA)
  - hyperglycemic hyperosmolar state (HHS)
  - insulin
  - intravenous fluids
  - potassium
  - sodium bicarbonate
- hyperkalemia
  - definition
  - peaked T waves
  - redistribution
  - reduce
  - “sine wave” pattern
  - stabilization
- hyponatremia
- hypoglycemia
  - definition
  - IV dextrose administration/oral carbohydrate administration
  - octreotide
  - sulfonylurea medications
  - sulfonylurea-induced hypoglycemia
- hypokalemia
- hyponatremia
- orthostatic hypotension (OH)
- thyroid storm
  - antithyroid medications
  - clinical presentation
  - diagnosis
  - management
  - passive cooling techniques
- venous blood gas (VBG)
  - bicarbonate
  - lactate



- partial pressure of carbon dioxide (PCO<sub>2</sub>)
- partial pressure of oxygen (PO<sub>2</sub>)
- pH
- End-of-life (EOL) care
- Endophthalmitis
- Endoscopic retrograde cholangiopancreatography (ERCP)
- Endoscopy
- Endotracheal intubation (ETI)
  - airway concerns
  - anticipated clinical course
  - oxygenation
  - ventilation
- Endotracheal tube (ETT) placement
  - chest x-ray
  - esophageal detector device
  - gastric insufflation
  - methods
  - objective confirmatory findings
  - pulse oximetry
  - qualitative colorimetric CO<sub>2</sub> capnometers
- End-tidal carbon dioxide (ETCO<sub>2</sub>)
  - capnography
  - chest compressions
  - endotracheal tube (ETT) patency
  - fluid resuscitation
- Environment
  - acute mountain sickness (AMS)
    - hallmark sign
    - Lake Louise consensus definition
    - prevention
    - symptoms
    - treatment
  - aggressive cooling
    - active cooling efforts
    - cold water immersion (CWI)
    - “Currie response”
    - dehydration
    - heat-related illness

- barotrauma injury
- CO poisoning
- decompression sickness (DCS)
- high-altitude cerebral edema (HACE)
  - Lake Louise consensus definition
  - treatment
- high-altitude pulmonary edema (HAPE)
- hypothermia, treatment of
- Lyme disease
- pulmonary overpressurization syndromes
- rocky mountain spotted fever (RMSF)
- smoke inhalation
  - systemic toxins
  - thermal burns
- thermoregulation
- tick-borne illnesses
- tularemia
- Epidural abscess
- Epiglottitis
  - differential diagnosis
  - evolution
  - invasive HiB disease
  - management
- Epinephrine
- Epoprostenol
- Erysipelas
- Erythema infectiosum
- Erythema migrans
- Erythema nodosum (EN)
  - causes
  - dermatologic conditions
  - incidence of
  - patients with
- Esmolol
- Esophageal rupture
- ETCO<sub>2</sub> *See (End-tidal carbon dioxide)*
- Ethylene glycol
- Etomidate

ETT placement *See (Endotracheal tube placement)*

Extension-type supracondylar fractures

Extracorporeal life support (ECLS)

Extracorporeal membrane oxygenation (ECMO)

- basic configuration

- indications

  - cardiogenic shock

  - hypercarbic respiratory failure

  - prolonged cardiac arrest

  - pulmonary embolism

  - toxic overdose

- physiology

- risks

- VA ECMO

- VV ECMO

Eyelid lacerations

## **F**

Facial bite wounds

Facial pain

Facial trauma

Familial hemiplegic migraine (FHM)

FAST *See (Focused assessment with sonography in trauma)*

Febrile nonhemolytic transfusion reactions (FNHTRs)

Femoral approach

Femoral nerve block

Fentanyl

Fetal fibronectin

FFP *See (Fresh frozen plasma)*

Fibrinolytic agent

Fifth metatarsal

Fitz-Hugh-Curtis syndrome (FHC)

Fluid responsiveness

Fluid resuscitation

- arterial waveform analysis

- end-tidal carbon dioxide

- inferior vena cava, ultrasound assessment

- intussusception

- major burns
- passive leg raise
- in trauma
- Fluid therapy
  - burns
  - classification
  - crystalloids vs. colloids
  - DKA/HHS
  - gastrointestinal (GI) losses
  - hyponatremia
  - IV fluid resuscitation
  - molecular weight and oncotic pressure
  - Parkland formula
  - rhabdomyolysis
  - trauma
  - types
- FNHTRs *See (Febrile nonhemolytic transfusion reactions)*
- Focused assessment with sonography in trauma (FAST)
- Fomepizole
- Forefoot
- Foreign bodies
  - ingestion, gastroenterology
  - wound care
- Fournier gangrene
- Fraction of inspired oxygen ( $\text{FiO}_2$ )
- Fractures
  - boxer's
    - metacarpal neck fracture and pattern of
    - radiographic characteristics
    - rotational deformity
  - calcaneus
  - carpal bone
  - extension-type supracondylar
  - hip
  - Jones
  - Maisonneuve
    - mechanism

- treatment of
  - pelvic
  - Pseudo-Jones/Dancer's
  - rib
  - scaphoid
  - scapula
  - supracondylar
    - compartment syndrome
    - CRITOE mnemonic
    - diagnosis
    - extension-type supracondylar fractures
    - types of
  - triquetrum
  - vertebral compression fractures (VCFs)
  - volar fractures
- Francisella tularensis*
- Fresh frozen plasma (FFP)
- Functional hemiparesis
- Fusobacterium necrophorum*

## G

- Gallbladder
  - cholecystitis
  - complete and full evaluation
  - duodenum, air in
  - gallbladder polyp
  - polyp
  - stone and edge artifact
  - wall echo shadow (WES) sign
- Gamekeeper's thumb
- Gastroenterology
  - acalculous cholecystitis
    - clinical presentation
    - complications
    - diagnosis and epidemiology
    - diagnostic imaging
    - pathophysiology
    - physical examination

- risk factors
- acute abdominal pain
  - early diagnosis
  - intravenous (IV) medications
  - policy statements
- acute appendicitis
- acute mesenteric ischemia (AMI)
  - acute mesenteric arterial occlusion
  - computed tomography angiogram (CTA)
  - D-dimer assay
  - management
  - mesenteric venous thrombosis
  - nonocclusive mesenteric ischemia
  - presentation
- acute pancreatitis
  - diagnosis
  - hazards of imaging
  - intervention and prognosis
  - presentation
- acute variceal hemorrhage (AVH)
  - consultation and disposition
  - presentation
  - stabilization
  - treatment
  - UGIB, etiology of
- altered mental status (AMS)
  - abdominal radiography
  - fecal occult blood
  - POC ultrasound
  - treatment
- anticipated bleeding
  - correction
  - laboratory abnormalities and treatment
- aortoenteric fistula (AEF)
  - definitive management
  - emergency department management
  - endovascular repair
  - of gastrointestinal bleeding

- primary and secondary
- ascending cholangitis
- biliary obstruction, causes
- Boerhaave syndrome
- caustic ingestions (CI)
  - acidic/alkaline
  - intentional ingestions
  - management
  - serious ingestions
- cecal volvulus
  - clinical presentation
  - imaging options
  - patient's history and physical examination
  - surgical management
  - symptoms
- chronic pancreatitis
  - abdominal pain
  - cigarette smoking
  - complications
  - ED management
  - harm reduction counseling
- coagulopathy
  - evaluation
  - treatment
- endoscopic retrograde cholangiopancreatography (ERCP)
- foreign body ingestion
- gallbladder
  - cholecystitis
  - complete and full evaluation
  - duodenum, air in
  - gallbladder polyp
  - stone and edge artifact
  - wall echo shadow (WES) sign
- inflammatory bowel disease (IBD)
  - abdominal series x-ray
  - complications
  - corticosteroids/immune modulators
  - opioids

- pathophysiology
- jaundice
  - definition
  - direct (conjugated) hyperbilirubinemia
  - hemolytic anemia
  - historical features
  - imaging modalities
  - initial testing
  - toxins
  - unconjugated hyperbilirubinemia
- peptic ulcer disease (PUD)
  - gastric outlet obstruction
  - perforation
  - UGIB
- percutaneous endoscopic gastrostomy (PEG) tube
- pregnancy
  - doxylamine succinate
  - fetal exposure estimation
  - imaging study
  - lifestyle and dietary changes
  - nausea and vomiting
  - pyridoxine hydrochloride
  - radiation exposure
- severe acute pancreatitis (SAP)
  - approach
  - Bedside Index of Severity of Acute Pancreatitis (BISAP) score
  - early identification and aggressive management
  - harmless acute pancreatitis (HAP) score
  - recognition of
- sigmoid volvulus
  - diagnosis
  - incidence
  - mortality
  - risk factors
  - treatment
- spontaneous bacterial peritonitis (SBP)
  - Bacterascites*
  - definition



- presumptive diagnosis
- primary and secondary prophylaxis
- signs and symptoms
- sources of infection
- treatment

Gastroesophageal reflux

GCSE *See (Generalized convulsive status epilepticus)*

Gelatins

Generalized convulsive status epilepticus (GCSE)

Genitourinary

- dialysis access

- dialysis disequilibrium syndrome (DDS)

- Fournier gangrene

- Paraphimosis

- phimosis

- priapism

  - management of

  - treatment of

  - type of

- pyelonephritis

- testicular torsion

- urethritis

  - diagnosis of

  - symptoms and management

  - treatment

Geriatrics

- abdominal pain

  - anchoring, avoiding

  - etiologies, high index of suspicion

  - imaging

  - symptoms

  - vital signs, exam, and labs

- acute coronary syndromes (ACSs)

  - diagnosis

  - ECG in

  - outcomes

  - presentation

  - symptoms

- treatment
- cognitive assessment
- communication quality and special considerations
- delirium
- dementia
- dizziness
  - causes of
  - head CT
  - history
  - physical exam
  - treatment
- elder abuse
- hip and vertebral compression fractures
- infection
- pain medications and procedural sedation
- patient advocacy
- polypharmacy
- trauma patients
- weakness
- Giant cell arteritis (GCA)
  - background
  - diagnosis
  - presentation
  - treatment
- Glasgow Coma Scale (GCS)
- Glucocorticoid therapy
- Graft
- Graft rejection
- Guillain-Barré syndrome (GBS)
- Gynecology *See (Obstetrics/gynecology)*

## **H**

- HACE *See (High-altitude cerebral edema)*
- Haemophilus influenza* type B
- Haldane effect
- Harmless acute pancreatitis (HAP) score
- HD *See (Hemodialysis)*
- Head CT (HCT)

- cerebrospinal fluid (CSF) shunts
- dizziness
- geriatric trauma patients
- lumbar puncture
- pediatric injuries
- subarachnoid hemorrhage (SAH)
- VP shunt

## Headache

- acute glaucoma
- acute mountain sickness (AMS)
- brain abscesses
- carbon monoxide toxicity
- carotid artery dissection
- cerebral venous sinus thrombosis (CVST)
- dialysis disequilibrium syndrome (DDS)
- giant cell arteritis
- idiopathic intracranial hypertension (IIH)
- intracranial pressure (ICP)
- intravenous immunoglobulin (IVIG)
- mucormycosis
- post-lumbar puncture
- syncope
- tick-borne illnesses

## Heart transplant patients

## HEENT

- acute vision loss
  - acute glaucoma
  - differential diagnosis
  - endophthalmitis
  - history
  - keratitis
  - physical exam
  - retinal artery occlusion
  - retinal detachment
  - uveitis
  - vitreous hemorrhage
- deep space neck infection
- children

- parapharyngeal abscesses (PPA)
- retropharyngeal (RPA)
- epiglottitis
  - differential diagnosis
  - evolution
  - invasive HiB disease
  - management
- epistaxis
- giant cell arteritis (GCA)
  - background
  - diagnosis
  - presentation
  - treatment
- herpes zoster ophthalmicus (HZO)
  - background
  - differential
  - presentation
  - tests
  - treatment
- Lemierre syndrome
  - characteristics
  - Fusobacterium necrophorum*
  - treatment
- Ludwig angina
- malignant otitis externa (MOE)
  - diagnosis
  - risk factor
  - treatment
  - workup
- odontogenic infection
  - exam
  - imaging
  - periapical abscess
  - treatment
- optic neuritis (ON)
- orbital cellulitis
- periorbital infections
- peritonsillar abscess (PTA)

- annual incidence
- antibiotic therapy
- complications
- disposition
- evaluation priorities
- imaging
- incision and drainage
- needle aspiration
- steroids
- strategies
- preseptal and postseptal cellulitis
- red eyes
  - acute angle-closure glaucoma
  - acute anterior uveitis
  - allergic conjunctivitis
  - bacterial conjunctivitis
  - blepharitis
  - episcleritis
  - hyperacute (gonococcal) conjunctivitis
  - inclusion (chlamydial) conjunctivitis
  - pterygium
  - scleritis
  - subconjunctival hemorrhage
  - superficial keratitis
  - viral conjunctivitis
- retrobulbar hematoma
  - adjunctive treatment
  - causes
  - decreased vision
  - decreased visual acuity
  - elevated intraocular pressure (IOP)
  - painful “tense” proptosis
  - proptosis
  - pupillary response
  - restricted/painful extraocular movements
- rhinocerebral mucormycosis
- tympanic membrane (TM)
- Hematology/oncology

- anticoagulant reversal
  - and hemorrhage
  - target-specific oral anticoagulants (TSOACs)
  - warfarin reversal
- hemolytic uremic syndrome (HUS)
  - causative agent
  - diagnosis of
  - treatment of
  - vs. TTP
- immune thrombocytopenia (ITP)
- leukostasis
- neutropenic fever
- thrombotic thrombocytopenic purpura (TTP)
  - cause of
  - Classic Pentad of
  - HUS vs.
  - treatment of
- tumor lysis syndrome (TLS)
  - Cairo-Bishop classification
  - diagnosis
  - kidney explosion
  - operational definition
  - pathogenesis of
  - step-by-step approach
  - symptoms of
  - treatment of metabolic abnormalities associated with
- warfarin
- Hemiplegic migraine
- Hemodialysis (HD)
  - alcohols
  - dialysis disequilibrium syndrome (DDS)
  - genitourinary
  - patients with tumor lysis syndrome (TLS)
  - salicylates toxicity
- Hemodynamic management
- Hemolytic transfusion reactions (HTRs)
- Hemolytic uremic syndrome (HUS)
  - causative agent

- diagnosis of
- diarrhea
- treatment of
- vs. TTP
- Hemoptysis
  - causes
  - evaluation
  - management
- Hemorrhage
  - acute variceal hemorrhage (AVH)
  - anticoagulants and
  - clinical practice
  - control
  - defined
  - fetomaternal
  - intracerebral hemorrhage (ICH)
  - nontraumatic subarachnoid hemorrhage
  - pelvic
  - postpartum complications
  - reverse life-threatening hemorrhage
  - shock, ATLS classification of
  - subconjunctival
  - vital signs
  - vitreous
- Hemorrhagic shock
- Hemostasis
- Heparin therapy
- Herpes zoster ophthalmicus (HZO)
- HFNC *See (High-flow nasal cannula)*
- HHS *See (Hyperglycemic hyperosmolar state)*
- High-altitude cerebral edema (HACE)
  - Lake Louise consensus definition
  - treatment
- High-altitude pulmonary edema (HAPE)
- High-flow nasal cannula (HFNC)
- Highly sensitive troponin assays
  - criteria
  - negative value

- positive value, etiologies of
- High-pressure injection injury
- High-resolution chest computed tomography (HRCT)
- HINTS test
- Hip
  - dislocation
    - classification
    - complications
    - hip joint
    - management
    - pelvis, anteroposterior (AP) film
  - fractures
- HIV *See (Human immunodeficiency virus)*
- HOCM *See (Hypertrophic cardiomyopathy)*
- HRCT *See (High-resolution chest computed tomography)*
- Human immunodeficiency virus (HIV)
  - coronary artery disease (CAD)
  - postexposure prophylaxis
  - tuberculosis
- Human trafficking
- HUS *See (Hemolytic uremic syndrome)*
- Hydroxyethyl starch (HES)
- Hyperbaric oxygen therapy
- Hyperemesis gravidarum
- Hyperglycemia
- Hyperglycemic hyperosmolar state (HHS)
- Hyperkalemia
  - Digoxin immune Fab
  - endocrine/metabolic
    - definition
    - peaked T waves
    - redistribution
    - reduce
    - “sine wave” pattern
    - stabilization
  - Wolff-Parkinson-White (WPW) syndrome
- Hyperleukocytosis
- Hypernatremia



Hyperosmolar hyperglycemic state (HHS)

Hypersensitivity

Hypertension

- idiopathic intracranial hypertension (IIH)

- intra-abdominal hypertension (IAH)

- intracerebral hemorrhage (ICH)

- management

- pulmonary hypertension (PH)

Hypertensive emergencies

Hyperthermia

Hypertonic saline

Hypertonic therapy

Hypertrophic cardiomyopathy (HOCM)

Hypoglycemia

- acute stroke

- endocrine/metabolic

  - definition

  - IV dextrose administration/oral carbohydrate administration

  - octreotide

  - sulfonylurea medications

  - sulfonylurea-induced hypoglycemia

- syncope

Hypokalemia

Hyponatremia

Hypoplastic left heart syndrome

Hypotension

- abdominal compartment syndrome (ACS) with

- cerebral perfusion pressure (CPP)

- definition

- multifocal atrial tachycardia (MAT)

- orthostatic hypotension (OH)

- peri-intubation

- postintubation

- with pulmonary hypertension (PH)

- toxic shock syndrome (TSS)

Hypothermia

Hypovolemia

Hypovolemic shock

Hypoxemia

Hypoxia

HZO *See (Herpes zoster ophthalmicus)*

## I

IAH *See (Intra-abdominal hypertension)*

IAP *See (Intra-abdominal pressure)*

IBD *See (Inflammatory bowel disease)*

ICH *See (Intracerebral hemorrhage)*

Idarucizumab

Idiopathic intracranial hypertension (IIH)

IIH *See (Idiopathic intracranial hypertension)*

IMI *See (Inferior myocardial infarction)*

Immune

- anaphylaxis

  - vs. angioedema

  - and epinephrine

- graft

- treacherous transplant toxicities

  - calcineurin inhibitors (CNIs)

  - mycophenolate

  - sirolimus

Immune thrombocytopenia (ITP)

Immunocompromised patients

IMV *See (Invasive mechanical ventilation)*

Incision and drainage (I&D)

- abscess

- antibiotics

- “loop drainage” technique

Infection

- geriatrics

- limping child

- postpartum complications

Infectious disease

- acute febrile illnesses

- acute retroviral syndrome (ARS)

  - clinical manifestation

  - HIV antibody tests

- symptoms of
- treatment of
- antibiotics
  - pneumonia
  - skin and soft tissue infections (SSTI)
  - urinary tract infections (UTI)
- bioterrorism agents
  - Bacillus anthracis*
  - Clostridium botulinum*
  - Francisella tularensis*
  - Variola major*
  - viral hemorrhagic fevers
  - Yersinia pestis*
- catheter-associated urinary tract infections (CAUTIs)
- chikungunya
- chronic obstructive pulmonary disease (COPD)
- diarrhea
- ebola virus disease (EVD)
- infective endocarditis (IE)
  - Duke criteria
  - mitral/aortic valve
  - presentation
  - prevalence
  - tricuspid valve
- influenza
- measles
- meningitis
  - cerebrospinal fluid analysis (CSF)
  - computed tomography (CT)
  - lumbar puncture
  - noninfectious etiologies of
  - physical examination
  - treatment
- middle east respiratory syndrome (MERS)
- necrotizing soft tissue infection (NSTI)
  - clinical presentation
  - diagnoses
  - history

- Laboratory Risk Indicator for Necrotizing Fasciitis score (LRINEC)
- progression and diffuse systemic involvement
- postexposure prophylaxis (PEP)
  - and follow-up
  - nonoccupational
  - occupational
- prevention measures
  - for common outbreaks
  - for rare epidemic outbreaks
  - routine
- staphylococcal toxic shock syndrome (TSS)
- syphilis
- systemic inflammatory response syndrome (SIRS)
- temperature measurement
- tuberculosis (TB)
- Infective endocarditis (IE)
  - Duke criteria
  - mitral/aortic valve
  - presentation
  - prevalence
  - prophylactic antibiotics
  - tricuspid valve
- Inferior myocardial infarction (IMI)
- Inferior vena cava (IVC)
  - evaluation
  - ultrasound assessment of
- Inflammation
  - keratitis
  - limping child
  - uveitis
- Inflammatory bowel disease (IBD)
  - abdominal series x-ray
  - complications
  - corticosteroids/immune modulators
  - opioids
  - pathophysiology
- Influenza
  - high-risk populations

- neuraminidase inhibitors
- oseltamivir
- peramivir
- treatment
- zanamivir
- INR *See (International normalized ratio)*
- Inspiratory positive airway pressure [iPAP]
- Inspiratory to expiratory ratio (I/E ratio)
- Institute for Healthcare Improvement (IHI)
- Interferon gamma release assays (IGRA)
- Internal defibrillation
- Internal jugular approach
- International normalized ratio (INR)
- Intimate partner violence (IPV)
- Intra-abdominal hypertension (IAH)
  - management algorithm
  - recognition and diagnosis
  - risk factors
  - trans-bladder pressure measurements
- Intra-abdominal pressure (IAP)
- Intracerebral hemorrhage (ICH)
- Intracranial hemorrhage (ICH)
- Intracranial pressure (ICP)
- Intramural hematoma (IMH)
- Intramuscular injection
- Intraosseous (IO) access
- Intraosseous line
- Intravenous (IV) access
  - central access
    - indication
    - limitations of
    - ideal location
  - intraosseous (IO) line
  - peripheral IV catheters
- Intravenous fluid resuscitation
- Intravenous immunoglobulin (IVIG)
- Intravenous lipid emulsion (ILE) therapy
- Intravenous opioids

Intussusception  
Invasive mechanical ventilation (IMV)  
IPV *See (Intimate partner violence)*  
Iron toxicity, five stages of  
Irrigation  
    bowel  
    traumatic wound  
Ischemic priapism  
Ischemic stroke  
    blood pressure measurement in  
    posterior circulation  
    transient ischemic attack (TIA)  
Isopropanol  
Isopropyl alcohol  
ITP *See (Immune thrombocytopenia)*  
Itra-abdominal pressure (IAP)  
IV access *See (Intravenous access)*

## **J**

Jaundice  
    definition  
    direct (conjugated) hyperbilirubinemia  
    hemolytic anemia  
    historical features  
    imaging modalities  
    initial testing  
    toxins  
    unconjugated hyperbilirubinemia  
Jones fracture  
J-point elevation

## **K**

Keratitis  
Ketamine  
Ketamine IM  
Kidney disease

## **L**

Laboratory Risk Indicator for Necrotizing Fasciitis score (LRINEC)

Laceration

ear

eyelid

intraoral

Large vessel vasculitis

Laryngomalacia

Laryngotracheobronchitis *See (Croup)*

LAST *See (Local anesthetic systemic toxicity)*

Late pregnancy

peripartum cardiomyopathy (PPCM)

vaginal bleeding

placenta previa

placental abruption

uterine rupture

vasa previa

Law suit

LBBB *See (Left bundle-branch block)*

Left bundle-branch block (LBBB)

Left upper quadrant (LUQ) abdominal view

Left ventricular hypertrophy (LVH)

Legg-Calvé-Perthes (LCP) disease

Lemierre syndrome

Leukocyte esterase (LE) test

Leukostasis

Limping child

infection

inflammation

injury

neoplastic

structural

Lipid sink theory *See (Intravenous lipid emulsion (ILE) therapy)*

Lipodermatosclerosis

Lisfranc injury

foot anatomy

imaging

initial assessment

management

- Local anesthetic systemic toxicity (LAST)
- Local anesthetics
- Loop diuretics
- Loop drainage technique
- Loperamide
- Low molecular weight heparin (LMWH)
- LPVS *See (Lung protective ventilation strategies)*
- Lumbar puncture
  - administering antibiotics
  - body mass index (BMI)
  - computed tomography (CT)
  - equipped and sterilization
  - fentanyl
  - need assess and performance
  - position
  - prevention
- Lumbar spine
- Lunate dislocation
- Lung protective ventilation strategies (LPVS)
- Lyme disease

## **M**

- MACE *See (Major adverse cardiac event)*
- Magnetic resonance angiography (MRA)
  - cervical vascular injuries
  - nontraumatic subarachnoid hemorrhage
- Magnetic resonance imaging (MRI)
  - back pain
  - Cauda equina syndrome (CES)
  - eclampsia
  - giant cell arteritis (GCA)
  - intracranial complications
  - necrotizing soft tissue infection (NTSI)
  - orthopedic injuries
  - trauma
  - vertebral compression fractures (VCF)
- Maisonneuve fracture
  - mechanism



- treatment of
- Major adverse cardiac event (MACE)
- Major burns
  - airway
  - fluid resuscitation
  - transfer
- Malignant otitis externa (MOE)
  - diagnosis
  - risk factor
  - treatment
  - workup
- Mammalian bites
  - clinical characteristics
  - MCP joint/fight bites
  - wound infection vs. nonbite wounds
- Mannitol
- Massive transfusion (MT)
  - ABC score
  - balanced resuscitation
  - definition
  - hemorrhagic shock
  - management of
  - rMTS
  - “survivor bias”
  - TASH score
- Massive transfusion protocols (MTPs) *See (Massive transfusion (MT))*
- MAT *See (Multifocal atrial tachycardia)*
- Measles
- Mechanical ventilation
- Meningitis
  - cerebrospinal fluid analysis (CSF)
  - computed tomography (CT)
  - lumbar puncture
  - noninfectious etiologies of
  - physical examination
  - treatment
- MERS *See (Middle east respiratory syndrome)*
- Metacarpal neck fractures

- Metacarpal phalangeal (MCP) joint
- Metaphysis/diaphysis—Jones fracture
- Methanol
- Methicillin-resistant *Staphylococcus aureus* (MRSA)
- Metoprolol
- Midazolam
- Middle east respiratory syndrome (MERS)
- Migraine, hemiplegic
- Minor abdominal trauma, in pregnancy
  - FAST exam
  - pregnancy
    - fetal monitoring, trauma
    - imaging
    - laboratory considerations
    - placental abruption/uterine rupture
    - primary and secondary survey
- Mitral/aortic valve infective endocarditis (IE)
- Mobitz type I AVB
- Mood disorders
- MRA *See (Magnetic resonance angiography)*
- Multifocal atrial tachycardia (MAT)
- Multiple sclerosis (MS)
- Muscarinic antagonism
- Myasthenia gravis (MG)
- Mycobacterium tuberculosis*
- Mycophenolate
- Mycophenolic acid (MPA)
- Myelopathy
- Myocardial infarction (MI)
  - right-sided ECG
  - ST-segment elevation myocardial infarctions (STEMIs) *See (ST-segment elevation myocardial infarctions)*

## N

- N*-acetyl-*p*-benzoquinone imine (NAPQI)
- Na/K ATPase pump
- Neck pain
- Necrotizing fasciitis

Necrotizing soft tissue infection (NSTI)

- clinical presentation

- diagnoses

- history

- Laboratory Risk Indicator for Necrotizing Fasciitis score (LRINEC)

- progression and diffuse systemic involvement

Neonatal resuscitation

- advanced resuscitation

- importance of warming

- initial resuscitation

Neonatal seizures

Neuraminidase inhibitors

Neurology

- acute stroke

  - cerebral neoplasm

  - functional hemiparesis

  - hemiplegic migraine

  - hypoglycemia

  - medical management

  - Todd paralysis

- botulism

- brain abscesses

- cerebral venous sinus thrombosis (CVST)

  - cause of

  - headache

  - heparin therapy

  - mechanisms

  - risk factors for

- cervical artery dissection (CAD)

  - carotid artery dissection

  - classification

  - clinical features

  - mechanical stressors

  - mimics

  - vertebral artery dissection

- Guillain-Barré syndrome (GBS)

- idiopathic intracranial hypertension (IIH)

- intracerebral hemorrhage (ICH)

- ischemic stroke
  - blood pressure measurement int
  - posterior circulation
- multiple sclerosis (MS)
- myasthenia gravis (MG)
- nontraumatic subarachnoid hemorrhage
  - computed tomography angiogram (CTA)
  - headache
  - magnetic resonance angiography (MRA)
  - management
  - noncontrast CT
- shunt malfunction
- status epilepticus
- transient ischemic attack (TIA)
- vertigo
- Neuropathy
- Neutropenic fever
- Nicardipine
- NIH Stroke Scale
- NIPPV *See (Noninvasive positive pressure ventilation)*
- Nitrates
- Nitroglycerin
- Nitroprusside
- Nitrous oxide (N<sub>2</sub>O)
- NIV *See (Noninvasive ventilation)*
- NOAC *See (Novel oral anticoagulant)*
- Nodules
- Nonaccidental trauma
- Noninvasive positive pressure ventilation (NIPPV)
- Noninvasive ventilation (NIV)
  - indications
    - asthma
    - blunt chest trauma
    - chronic obstructive pulmonary disease
    - congestive heart failure
    - pneumonia/immunocompromised patients
    - preoxygenation
  - modes

- patient selection
- Nonischemic priapism
- Nonoccupational postexposure prophylaxis
- Nonsteroidal anti-inflammatory medications (NSAIDs)
  - pain medications
  - perforation
- Nontrauma
  - acute compartment syndrome (ACS)
  - back pain
  - cauda equina syndrome (CES)
  - rhabdomyolysis
  - rheumatoid arthritis
  - septic arthritis
  - spondyloarthropathies
- Nontraumatic subarachnoid hemorrhage
  - computed tomography angiogram (CTA)
  - headache
  - magnetic resonance angiography (MRA)
  - management
  - noncontrast CT
- Normal vital signs, in pregnancy
  - blood pressure
  - fetal heart tones
  - heart rate
  - respiratory rate/oxygen saturation
- Norwood
- Novel oral anticoagulant (NOAC)
- NSTI *See (Necrotizing soft tissue infection)*
- Nuchal cords

## O

- Obsessive compulsive personality disorder (OCPD)
- Obstetrics/gynecology
  - anti-D
  - cords
  - early pregnancy
  - eclampsia
  - late pregnancy, vaginal bleeding

- placenta previa
- placental abruption
- uterine rupture
- vasa previa
- minor abdominal trauma, pregnancy
  - FAST exam
  - fetal monitoring, trauma
  - imaging
  - laboratory considerations
  - placental abruption/uterine rupture
  - primary and secondary survey
- normal vital signs, pregnancy
  - blood pressure
  - fetal heart tones
  - heart rate
  - respiratory rate/oxygen saturation
- ovarian torsion
  - bedside exam
  - classic
  - CT
  - ultrasound
- perimortem cesarean sections (PMCS)
- postpartum complications
  - fever and infection
  - hemorrhage
  - neuropathy
  - peripartum cardiomyopathy
  - preeclampsia/eclampsia
  - vulvar edema
- preterm labor
- shortness of breath
  - pregnancy
  - without peripartum cardiomyopathy (PPCM)
- Obstructive shock
- Occupational postexposure prophylaxis
- OCPD See (*Obsessive compulsive personality disorder*)
- Odd disorders
- Odontogenic infection

- exam
- imaging
- periapical abscess
- treatment

OH *See (Orthostatic hypotension)*

Olanzapine

Older adults *See also (Geriatrics)*

- abdominal pain in
- acute coronary syndromes (ACS)
- altered mental status in
- cognitive assessment
- communication
- communication quality and special considerations
- dizziness
- pain medications and procedural sedation
- patient advocacy
- weakness

Oncology *See (Hematology/oncology)*

ONSF *See (Optic nerve sheath fenestration)*

Opioid

Optic nerve sheath fenestration (ONSF)

Optic neuritis

Oral anticoagulant medications

- novel oral anticoagulant (NOAC) medication
- target-specific oral anticoagulants (TSOACs)
- vitamin K antagonist (VKA)

Oral temperature measurement

Orbital compartment syndrome

Organic brain syndrome

Organic psychosis

Organophosphate (OP)

Oropharynx, colonization of

Orthology/trauma

- achilles tendon
- ankle-brachial index (ABI)
- antiplatelet medications
- boxer's fracture
  - metacarpal neck fracture and

- pattern of
  - radiographic characteristics
  - rotational deformity
- calcaneus fractures
- cervical vascular injury
- chest tube placement
  - complications
  - drainage
  - indications
  - point-of-care ultrasound
  - radiography
- electrical injuries
- fluid resuscitation
- focused assessment with sonography in trauma (FAST)
- Gamekeeper's thumb
- geriatric patients
- geriatrics
- hemorrhage
  - clinical practice
  - defined
  - shock, ATLS classification of
  - vital signs
- high-pressure injection injury
- hip dislocation
- imaging indications
  - blunt trauma
  - penetrating trauma
- interventional radiology (IR)
- intimate partner violence and human trafficking
- intravenous (IV) access
- Maisonneuve fracture
- massive transfusion (MT)
- metaphysis/diaphysis-Jones fracture
- novel anticoagulant (NOAC)
- packed red blood cells
- pediatrics
- pelvic fractures
- penetrating neck injuries



- computed tomography angiogram (CTA)
- evaluation and management
- hard and soft signs
- patient with CTA and nonconcerning trajectory
- workup
- wound, superficial/deep
- zones
- perilunate and lunate dislocations
- pregnant women
- proximal diaphysis—stress fractures
- pseudo-Jones/Dancer's fracture
- rib fractures
- scaphoid fracture
- scapholunate dislocation/dissociation (SLD)
  - AP radiograph
  - mechanism
  - presentation
  - scaphoid tilt test
- scapula fractures
- spinal immobilization
- supracondylar fractures
- Thomas A. Swift Electric Rifle (TASER)
  - management
  - mode
  - predisposing/associated conditions
  - removal
  - serious injury, failure
- thoracotomy
  - aortic cross-clamping, indications and technique
  - benefit of
  - cardiac massage
  - complications
  - contraindications
  - damage control
  - incision making
  - indications
  - internal defibrillation
  - organ exposure

- pericardiotomy, indications and technique
- resuscitation process
- traumatic brain injury (TBI)
  - adequate oxygenation and cerebral perfusion pressure
  - adequate sedation
  - classifying and defining
  - hypotension and ensuring venous drainage
  - osmotic therapy
  - oxygen saturation and ventilation
  - seizure prophylaxis
- vascular injury
  - CT angiogram
  - evolution
  - hard and soft signs
  - hemorrhage control
  - proximity wounds
  - surgical exploration
- volar fractures
- Orthopedic injuries
- Orthostatic hypotension (OH)
- Oseltamivir (Tamiflu®)
- Osmotic therapy
- Otitis media (OM)
- Outpatient referral services
- Ovarian torsion
  - bedside exam
  - classic
  - CT
  - ultrasound
- Overcirculation

## **P**

- Packed red blood cells
- Pain
- Panic disorder
- Papular urticaria
- Paracentesis
  - hemorrhagic complications

- indications
- mechanical complications
- nonhemorrhagic complications
- technique
- Paralysis
  - bilateral vocal cord
  - rapid sequence intubation (RSI)
- Todd
- Paraphimosis
- Partial pressure of carbon dioxide (PaCO<sub>2</sub>)
- Partitioning theory *See (Intravenous lipid emulsion (ILE) therapy)*
- Passive leg raise (PLR)
- PCC *See (Prothrombin complex concentrate)*
- PE *See (Pulmonary embolism)*
- PEA *See (Pulseless electrical activity)*
- Pediatric Emergency Applied Research Network (PECARN)
- Pediatrics
  - abuse
  - airway
  - apparent life-threatening event (ALTE)
    - defined
    - dilemma
    - evaluation
    - gastroesophageal reflux
    - imaging
  - appendicitis
    - imaging
    - treatment
  - burns
  - concussion
  - crying
  - cyanotic postoperative cardiac patients
    - bidirectional Glenn
    - Hemi-Fontan
    - hypoplastic left heart syndrome
    - overcirculation
    - pulse oxygen
    - repair

- deep space neck infections
- diabetic ketoacidosis (DKA)
- ECG
- head CT (HCT)
- intussusception
- limping child
  - infection
  - inflammation
  - injury
  - neoplastic
  - structural
- neonatal resuscitation
  - advanced resuscitation
  - importance of warming
  - initial resuscitation
- procedural sedation
  - ketamine
  - midazolam
  - nitrous oxide
  - preparation
  - propofol
  - sedative-hypnotics and analgesic agents
- pyloric stenosis
  - diagnosis of
  - lab investigations
  - physical exam
  - pyloric stenosis
  - ultrasound imaging
- resuscitation
- sick neonate
  - injuries
  - multiple metabolic syndromes
  - neonatal seizures
  - respiratory illness
  - vomiting
- stridor
- urinary tract infection (UTI)
  - causes

- culture
- delayed treatment
- diagnosis and management
- leukocyte esterase (LE)
- nitrite test
- SPA and bladder catheterization
- urinalysis components, sensitivity and specificity
- PEEP *See (Positive end-expiratory pressure)*
- Pelvic fractures
- Pelvic hemorrhage
- Pelvic inflammatory disease (PID)
  - considerations
  - diagnosis
  - treatment
- Penetrating neck injuries
  - computed tomography angiogram (CTA)
  - evaluation and management
  - hard and soft signs
  - patient with CTA and nonconcerning trajectory
  - workup
  - wound, superficial/deep
  - zones
- Penetrating trauma
- PEP *See (Postexposure prophylaxis)*
- Peptic ulcer disease (PUD)
  - gastric outlet obstruction
  - perforation
  - UGIB
- Peramivir (Rapivab®)
- Percentage of glottis opening (POGO) score
- Percutaneous endoscopic gastrostomy (PEG) tube
- Percutaneous sutures
- Pericardial effusion
- Pericardiotomy
- Peri-intubation hypotension
- Perilunate dislocation
- Perimortem cesarean sections (PMCS)
- Peripartum cardiomyopathy (PPCM)

- Peripheral IV catheters
- Peritonsillar abscess (PTA)
  - complications
  - imaging
  - management
    - annual incidence
    - antibiotic therapy
    - disposition
    - evaluation priorities
    - incision and drainage
    - needle aspiration
    - steroids
    - strategies
- Personality disorders
  - anxious disorders
  - definition
  - dramatic disorders
  - odd disorders
  - psychiatric patient
- PH *See (Pulmonary hypertension)*
- Phimosis
- Physostigmine
- PID *See (Pelvic inflammatory disease)*
- Pigtail catheters
- PIH *See (Postintubation hypotension)*
- Placenta previa
- Placental abruption
- Plantar puncture
  - classification
  - risk of infection
- Plateau pressure ( $P_{plat}$ )
- PMCS *See (Perimortem cesarean sections)*
- Pneumonia
  - antibiotics
  - chronic obstructive pulmonary disease (COPD)
  - NIV
- Pneumonitis
- Pneumothoraces

- Pneumothorax
  - spontaneous
  - tension
    - chest pain
    - needle decompression
- Point-of-care ultrasound (POCUS)
- Polypharmacy
- Positive end-expiratory pressure (PEEP)
- Positive pressure ventilation (PPV)
  - acute pulmonary edema
  - bag-valve mask (BVM)
  - refractory shock
- Postanginal sepsis *See (Lemierre syndrome)*
- Postexposure prophylaxis (PEP)
  - and follow-up
  - nonoccupational
  - occupational
- Postintubation hypotension (PIH)
  - complications
  - identify and intervene
  - induction agents
  - insufficient venous return
  - new/evolving medical problems
  - predict and prevention
  - predisposing factors
- Postpartum complications
  - fever and infection
  - hemorrhage
  - neuropathy
  - peripartum cardiomyopathy
  - preeclampsia/eclampsia
  - vulvar edema
- Postpartum eclampsia
- Postpartum hemorrhage
- PP *See (Plateau pressure)*
- PPCM *See (Peripartum cardiomyopathy)*
- Pralidoxime (2-PAM)
- Pregnancy

- doxylamine succinate
- fetal exposure estimation
- imaging study
- lifestyle and dietary changes
- minor abdominal trauma in
  - Fast exam
  - fetal monitoring, trauma
  - imaging
  - laboratory considerations
  - placental abruption/uterine rupture
  - primary and secondary survey
- nausea and vomiting
- normal vital signs in
  - blood pressure
  - fetal heart tones
  - heart rate
  - respiratory rate/oxygen saturation
- potential catastrophes from worried well
  - $\beta$ -HCG test
  - CBC and CMP
  - Rh typing
  - ultrasound
  - vital signs
- pyridoxine hydrochloride
- radiation exposure
- shortness of breath
  - diagnosis
  - pathophysiology
  - presentation
  - treatment

#### Preoxygenation

- “delayed sequence intubation,” approaches
- desaturation levels
- method selection
- with NIPPV
- patient position
- patient’s normal respiratory pattern
- pre-and peri-intubation periods



Prescription cascade

Preterm labor

- defined

- diagnosis

- fetal fibronectin test

- risk factors

- sound understanding

Priapism

- management of

- treatment of

- type of

Prinzmetal angina

Procainamide

Procedural sedation

- anticipate adverse events

- available resources

- definition

- older adults

- patient assessment

- pediatric

  - ketamine

  - midazolam

  - nitrous oxide

  - preparation

  - propofol

  - sedative-hypnotics and analgesic agents

- pharmacologic selection

Procedures/skills/anesth

- arthrocentesis

- capnography

  - confirming placement

  - for CPR

  - for procedures

  - respiratory distress evaluation

- central line placement

  - avoiding arterial injury

  - site selection

- intraosseous (IO) access

- lumbar puncture
  - administering antibiotics
  - body mass index (BMI)
  - computed tomography (CT)
  - equipped and sterilization
  - fentanyl
  - need assess and performance
  - position
  - prevention
- paracentesis
  - hemorrhagic complications
  - indications
  - mechanical complications
  - nonhemorrhagic complications
  - technique
- procedural sedation
  - anticipate adverse events
  - available resources
  - defined
  - patient assessment
  - pharmacologic selection
- spontaneous pneumothorax
- tension pneumothorax, needle decompression
- transfusion
  - active bleeding
  - anemia
  - numerical thresholds
  - Transfusion Requirements in Critical Care trial
- transfusion reactions
  - allergic transfusion reactions (ATRs)
  - febrile nonhemolytic transfusion reactions (FNHTRs)
  - hemolytic transfusion reactions (HTRs)
  - transfusion-associated circulatory overload (TACO)
  - transfusion-associated graft *versus* host disease
  - transfusion-associated sepsis (TAS)
  - transfusion-related acute lung injury (TRALI)
- ultrasound-guided regional anesthesia
- VP shunt placemen

- cerebrospinal fluid (CSF) analysis
- complications
- imaging
- indications
- malfunction
- primary reason
- Prolapsed cord
- Prolonged QT syndrome
- Prophylactic antibiotics
  - deep structure
  - for dental procedures
  - environmental exposures
  - infective endocarditis (IE)
  - intraoral lacerations
  - mammalian bite wounds
  - patient factors
  - puncture wounds
  - wound factors
- Propofol
- Propranolol
- Protease inhibitor (PI)
- Prothrombin complex concentrate (PCC)
- Proton pump inhibitors (PPIs)
- Proximal diaphysis—stress fractures
- Proximal fibula fracture *See (Maisonneuve fracture)*
- Proximal fibular pain
  - historical complaint
  - Maisonneuve fracture with
- Pseudohyperkalemia
- Pseudo-Jones/Dancer's fracture
- Psychiatric disorder
  - affective disorders
  - anxiety
    - anxious state
    - evaluation
    - medical conditions with
    - treatment
  - delirium

- diagnosis
  - etiologies of
  - treatment
- drug-seeking behavior
- personality disorders
- psychosis
- restraint with restraints
- substance abuse
  - harm reduction
  - intervention
  - screening
  - treatment
- suicide risk
- Psychosis
- PTA *See (Peritonsillar abscess)*
- PUD *See (Peptic ulcer disease)*
- Pulmonary edema
  - aggressive nitroglycerin usage
  - positive pressure ventilation (PPV)
  - “tank overload”
  - treatment adjuncts in
- Pulmonary embolism (PE)
  - chest pain
  - clinical symptoms
  - massive
  - in pregnancy
  - thoracic
    - diagnosis and treatment
    - risk stratify patients with
    - thrombolytic therapy
- Pulmonary embolism severity index (PESI)
- Pulmonary hypertension (PH)
  - causes
  - decompensated patients, resuscitation of
  - hypoxia
  - sepsis
  - supraventricular tachyarrhythmias
  - vasodilator medications, abrupt discontinuation

- WHO Group 1, vasodilators
- World Health Organization classification
- Pulmonary overpressurization syndromes
- Pulse oximetry
- Pulseless electrical activity (PEA)
  - “3 and 3 rule,”
  - Hs and TS
  - QRS
  - sonographic algorithm
- Pyelonephritis
- Pyloric stenosis
  - diagnosis of
  - lab investigations
  - physical exam
  - pyloric stenosis
  - ultrasound imaging

## Q

- Quick Sequential Organ Failure Assessment score

## R

- Radial collateral ligament (RCL)
- Radiation therapy (RT)
- Rapid sequence intubation (RSI)
  - paralysis
  - premedication
  - preparation
  - sedation
- Rapid ventricular response (RVR), atrial fibrillation with
  - amiodarone
  - beta-adrenergic blockers
  - calcium channel blockers (CCBs)
  - digoxin
  - Wolff-Parkinson-White patients
- Rashes
  - classic pediatric rashes
  - classic tick-borne illnesses
- RASS See (*Richmond Agitation Sedation Scale*)

REBOA *See (Resuscitative endovascular balloon occlusion of the aorta)*

Recombinant factor VIIa (rFVIIa)

Rectal temperature measurement

Red blood cell transfusion

Red eye

- acute angle-closure glaucoma
- acute anterior uveitis
- allergic conjunctivitis
- bacterial conjunctivitis
- blepharitis
- episcleritis
- hyperacute (gonococcal) conjunctivitis
- inclusion (chlamydial) conjunctivitis
- pterygium
- scleritis
- subconjunctival hemorrhage
- superficial keratitis
- viral conjunctivitis

Refractory hypoxemia

- acute respiratory distress syndrome (ARDS)
- "DOPES" mnemonic
- endotracheal tube (ETT)
- "MASH" approach

Refractory shock

Respiratory illness

Respiratory rate

Respiratory syncytial virus (RSV)

Restraints

Resuscitation

- balanced
- fluid
  - arterial waveform analysis
  - end-tidal carbon dioxide
  - inferior vena cava, ultrasound assessment
  - intussusception
  - major burns
  - passive leg raise
  - in trauma

- intravenous fluid
- neonatal
  - advanced resuscitation
  - importance of warming
  - initial resuscitation
- Resuscitative endovascular balloon occlusion of the aorta (REBOA)
- Resuscitative thoracotomy
  - complications
  - initial approach
  - objective signs of life
  - outcomes
  - survival rate
- Retinal artery occlusion
- Retinal detachment
- Retrobulbar hematoma
  - adjunctive treatment
  - causes
  - decreased vision
  - decreased visual acuity
  - elevated intraocular pressure (IOP)
  - painful “tense” proptosis
  - proptosis
  - pupillary response
  - restricted/painful extraocular movements
- Retro-orbital hematoma
- Return of spontaneous circulation (ROSC)
- Revised Massive Transfusion Score (rMTS)
- Rhabdomyolysis
- Rheumatoid arthritis (RA)
- Rib fractures
- Richmond Agitation Sedation Scale (RASS)
- Right upper quadrant (RUQ) abdominal view
- Right ventricle (RV) failure
- Right ventricular myocardial infarction (RVMI)
- Risperidone (Risperdal)
- Rocky mountain spotted fever (RMSF)
- Rocuronium
- ROSC *See (Return of spontaneous circulation)*

Roseola  
Rotational deformity  
RSI *See (Rapid sequence intubation)*  
Rubbing alcohol  
Rubella

## S

SADPERSONS scale  
Salicylates

- endotracheal intubation and mechanical ventilation
- hemodialysis
- intravenous fluid resuscitation
- management
- medical applications
- metabolic acidosis
- signs and symptoms
- toxicity

*SAP See (Severe acute pancreatitis)*  
Sarcoidosis  
*SBP See (Spontaneous bacterial peritonitis)*  
Scaphoid fracture  
Scaphoid tilt test  
Scapholunate dislocation/dissociation (SLD)

- AP radiograph
- mechanism
- presentation
- scaphoid tilt test

Scapula fractures  
Scarletina/scarlet fever  
*SCD See (Sickle cell disease)*  
SCORTEN system  
SedationSedative-hypnotics

- adequate
- analgo-sedation
- chemical
- dexmedetomidine
- ketamine
- procedural



- anticipate adverse events
- available resources
- definition
- ketamine
- midazolam
- nitrous oxide
- older adults
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*Variola major*

Vasa previa

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VCFs *See (Vertebral compression fractures)*

Venoarterial (VV) ECMO

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- lactate

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- partial pressure of oxygen (PO<sub>2</sub>)

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## **W**

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  - antivenin Crotalidae Polyvalent Immune Fab (Ovine)
  - coral snake venom
  - pit viper bites
- systemic loxoscelism
- traumatic wound irrigation
  - irrigation
  - personal protection
  - preprocedural preparation
  - scrubbing
  - skin preparation
- wound management

## **X**

Xanthochromia

X-ray

## **Y**

*Yersinia pestis*

## **Z**

Zanamivir (Relenza®)

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